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MENIERE'S SYNDROME.*

DR. HANS BRUNNER, Chicago.

A large amount of work has been done on the pathology and treatment of Ménière's syndrome, although it seems that the results are not proportionate to the effort applied. This is to be expected, since almost every author has his own conception of Ménière's syndrome, if one is to judge from the different diagnostic criteria presented.

DIAGNOSIS.

A distinction must be drawn between the symptoms of the acute attack and those of the intervening periods. The acute spells are frequently ushered in by intense tinnitus, the patient often perceiving a whistling high tone. The tinnitus is followed by vertigo and diminution of hearing. The dizzy spells may be slight, moderate or severe and, accordingly, are accompanied by spontaneous nystagmus and vasomotor symptoms of different degrees. In severe spells the nystagmus reaches a third degree and is most often directed toward the deaf side; less frequently to the healthy side. Thornvall[57] emphasizes that the nystagmus during the spell is as a rule rotatory. DeKleyn and Versteegh[41] found that the nystagmus which is towards the deaf side during the attack turns to the other side when the attack has ceased. The vasomotor symptoms also depend on the intensity of the spell, increasing from pallor of the face to vomiting, with difficulty in vision to unconsciousness during the attacks but never any convulsive movements. Naturally it is rarely possible to perform

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*From the Department of Laryngology, Rhinology and Otology, and the Research and Educational Hospital of the University of Illinois College of Medicine.

exact functional ear tests during the spell; however, in one of my cases the following findings were obtained:

A girl, age 26 years, had a dry perforation in the anterior inferior quadrant of the left drum. The right drum was retracted and white. The right malleus could not be moved by Siegle's test. Accentuated whispered voice was heard ad concham on the right side, and 2 metres on the left. Weber was not lateralized. Schwabach was prolonged on both sides. Rinné was negative on both sides. C was not heard, and the watch was audible via both mastoids. High tones were a little diminished on the left, and markedly diminished on the right side. Gellé was negative on the right. There was tinnitus, some dizziness and vomiting; a spontaneous rotatory nystagmus of third degree to the left side was diminished by bending the head backwards. If the head was turned toward the left shoulder she fell to the right and forward, and if the head was turned to the right shoulder she fell to the right and backwards. After irrigating the right ear with cold water, the spontaneous nystagmus was slightly increased; after irrigating the right ear with hot water, no nystagmus to the right could be elicited. After irrigating the left ear with cold water, a nystagmus to the right appeared for a few seconds but was soon suppressed by the spontaneous nystagmus to the left. Similar results were obtained with the turning chair. Eleven days later, she had only a first degree nystagmus to the left. There was no vertigo but the tinnitus still remained. There was a definite diminution of the excitability of the right labyrinth for the caloric test, as well as for the turning test.

In this case the spell was undoubtedly produced by the right ear, which was deaf during the attack and did not respond to the caloric and turning tests; however, the spontaneous nystagmus was to the left. It is noteworthy that no other attacks occurred for many years. The treatment was conservative, consisting of Faradization of the inner ear and inflations of acetic ether into the middle ear.

Summarizing, we may say that Ménière's attack consists of: *1.* cochlear symptoms (tinnitus, deafness); *2.* labyrinthine symptoms (dizziness, spontaneous nystagmus, diminution of the excitability of one labyrinth); and *3.* general vasomotor symptoms. In order to avoid confusion in the diagnosis, it is important to keep this triad of symptoms in mind. It is possible, of course, that one or another of the mentioned symptoms may be missing in any one single spell. In that case a further observation of the patient is necessary to determine whether or not the entire triad occurs. The diagnosis of Ménière's attack is justified only when the triad is present.

Between the attacks there is often to be found a retracted, fibrous or atrophic drum, with or without scars after perforation (Brunner,[7] Mygind and Dederding,[48] and Eisenberg[24]); however, the drum may be normal. Tinnitus is present as a rule and has a different intensity and exacerbates either before or during an attack. Of 93 patients of Crowe,[18]

eight had no tinnitus at any time. If this disease is of short duration the hearing may be normal. In chronic cases there is, as a rule, progressive deafness, which, according to Crowe,[18] has the peculiar characteristic of fluctuation. The deafness is a conductive lesion with distinct bass quality (Thornvall,[60] Mygind and Dederding[49]), or a conductive and perceptive lesion, or a pure inner lesion (Crowe). If there is impairment of hearing on both sides, it is usually more marked on one side.

The Ménière's attack usually produces an acute increase of deafness which outlasts the attack. Very interesting, although difficult to understand, is the fact that an occasional attack is followed by a definite improvement of hearing. v. Frankl-Hochwart[26] mentions such a case. Other observers have had the same experience. Dizziness and spontaneous nystagmus between the attacks may or may not be present. At any rate, both symptoms are not very marked. The examination of the labyrinth rarely reveals a hyperexcitability (Kobrak,[40] DeKleyn and Versteegh[41]), or normal findings (Thornvall,[60] Furstenberg, Lashmet and Lathrop,[27] and Crowe[18]). If in the latter case, more precise methods for testing are employed it will usually be found that the labyrinth on one side can be stimulated more easily than that on the other side (DeKleyn and Versteegh). Thornvall, Mygind and Dederding[50] emphasize that a reduction of the labyrinthine function is "extremely rare." I would rather say that the finding is not common. In agreement with Dandy,[19] Eisenberg,[24] Crowe,[18] Hallpike and Cairns,[33] I do not believe it is "extremely rare." Failure of the one labyrinth to react has rarely been observed. The differences in the findings are dependent on the extent to which the disease has progressed.

Vogel[68] observed, in cases of Ménière's syndrome, a phenomenon which he calls "Nystagmusbereitschaft." It means that in these patients a nystagmus toward one side can be elicited more easily than toward the other, independent of the method of stimulation used. In other words, a nystagmus to the right can be elicited more easily than to the left, whether using cold water in the left ear or hot water in the right. The finding of Vogel was confirmed by German,[20] but seen only occasionally by DeKleyn and Versteegh.

Mygind and Dederding often discovered headache, tender edema of the skin, nycturia and low blood pressure in their

cases. Crowe, however, found that the blood pressure as a rule was normal.

DIFFERENTIAL DIAGNOSIS.

The syndrome of Ménière is well defined if one keeps in mind that it consists of spells and of a chronic progressive atrophy of the inner ear; that the attacks essentially consist of labyrinthine and cochlear symptoms, and that the atrophy involves the cochlear more than the labyrinthine division of the inner ear; nevertheless, errors in the diagnosis are possible, even as far as the spells are concerned.

Unfortunately, we do not often have the opportunity to observe the patient during an attack; consequently, the diagnosis is entirely based on the history. Since this is usually unreliable, it permits only a tentative diagnosis. We are able to make a positive diagnosis if the spontaneous nystagmus is observed during the attack. This statement, in all probability, seems too exacting; however, if one has seen many cases of Ménière's syndrome and has observed them for a long time, he knows that hysteria may offer, occasionally, the subjective symptoms of a Ménière's attack. Famous is the case of v. Frankl-Hochwart[26] concerning a woman who nursed her husband during his spell. When the husband was cured from his actual Ménière's syndrome, the wife continued the spells in a very detailed, although hysterical, manner. Naturally, in these cases the objective symptoms of Ménière's syndrome, such as spontaneous nystagmus and progressive atrophy of the inner ear, are absent.

If there is no chance to observe a spontaneous nystagmus during a spell, we have one possibility to corroborate the diagnosis from the history of the patient. It is an old observation that a patient who suffers from a nystagmus of high degree prefers to lie on a certain side, since in that position the nystagmus and, consequently, the dizziness decreases. If we take, for instance, a patient who has an acute attack of labyrinthine nystagmus of third degree to the right, we know that the nystagmus and the dizziness definitely decrease when the patient looks toward his left side, while both symptoms increase when the patient looks toward his right side. When such a patient lies on his right side, the eyes automatically deviate to the left. Consequently, the patient's nystagmus

and dizziness definitely decrease when the patient lies on his right side. Therefore, if during his spell the dizziness decreases markedly in lying on a certain side of his body, and markedly increases in lying on the opposite side, we can draw the conclusion that the patient had a spontaneous nystagmus of high degree during his spell.

However, it must be remembered that not all vertigo complained of by the patient is of labyrinthine origin. From the clinical point of view, only the so-called "turning dizziness" and the "tactile dizziness" refer definitely to the labyrinth. Consequently, only these two kinds of vertigo characterize the syndrome of Ménière.*

Ménière's syndrome must not be confused with "Pseudo-Ménière's disease," a diagnosis which was introduced by v. Frankl-Hochwart many years ago, and recently revived by Dandy.[20] This re-emphasis would have preferably been omitted, since this word seems to increase the confusion about Ménière's syndrome. Practically every writer who uses this expression means another thing. v. Frankl-Hochwart refers to "Pseudo-Ménière" as spells of dizziness, tinnitus and vomiting, which appear as an aura of epileptical or hysterical attacks. It is obvious that in that meaning "Pseudo-Ménière" is not an actual diagnosis but an excuse for a wrong diagnosis.

Dandy states that in "Pseudo-Ménière's disease," the attacks are exactly like those of Ménière's syndrome, but there is never unilateral deafness. These attacks come on without any change in hearing. We cannot agree with Dandy. The attacks described by him are either actually Ménière's disease, which would reveal unilateral diminution of hearing, if the patient were observed for a longer period of time, or they are attacks of vertigo associated with cerebellar instead of cochlear symptoms. Consequently, these are not Ménière's attacks, although originally Ménière included the cerebellar symptoms in the syndrome described by him. Such spells, which never produce either tinnitus or deafness, may be found in syphilis, essential hypertension, disseminated sclerosis, cerebellopontine angle tumors, etc. In calling these attacks "Pseudo-Ménière," one takes the risk of masking a very serious ailment, as can be shown by the following case:

*As far as the concept of vertigo is concerned, there is a great difference between the psychologic point of view in general and the otological point of view as seen by comparison of the articles of W. R. Brain[65] and H. Brunner.[66]

A young man, age 21 years, became ill with headache and vomiting in June, 1928. On Dec. 24, 1928, he developed vertigo, accompanied by headache, vomiting and abasia. In January, 1929, there was found a slight paresis of the left abducens muscle and pastpointing in the finger-nose test. The examination of the eye did not reveal any pathologic change; the ears showed bilateral scarring of both tympanic membranes following perforation. No abnormal response was obtained on functional testing of the ears. The diagnosis of "Pseudo-Ménière's attack" was made.

In May, 1929, he again became ill with vertigo, headache, vomiting and neck pain. The symptoms disappeared following bed rest. In November, 1929, there was a recurrence, accompanied for the first time by unconsciousness. No convulsions, biting of the tongue or incontinence was present. In December, 1929, he noticed paresis and paresthesia in the right arm and left leg. He felt cold in the right half of the body, which showed a considerable cyanosis. Again all symptoms improved. In May, 1930, he again became ill with the dizzy spells, particularly when he was lying on his left side. The dizzy spells were accompanied at that time by headache, motor aphasia and convulsions in the legs. On the left, the corneal reflex was missing. The forehead was hyperesthetic and hyperalgesic, and the reflexes of the skin were diminished. There was spastic paresis on the right side, but no cerebellar symptoms could be found. The left pupil did not react as well as the right one, and there was definite choking of discs. The examination of the ears revealed impairment of hearing of very slight degree on both sides. There was a fine nystagmus (I) to both sides. The nystagmus to the right was increased by quick head movements and there was hyperexcitability of both labyrinths, more on the left than on the right, although bilateral calorization did not elicit nystagmus.

A diagnosis of tumor of the frontal lobe was made, but at operation no tumor was found. After the operation, patient developed a marked tenderness of the neck and of both mastoids, a horizontal nystagmus to both sides, more to the right than to the left, a flaccid paresis of the right arm, and a spastic paresis of the right leg. Consequently, the cerebellum was exposed on June 26, 1930, but during the procedure a severe collapse occurred and the operation had to be stopped. One month later, the patient died, and the autopsy revealed a chronic progressive tuberculosis of the lungs with a cavity as big as a walnut in the left upper lobe, many tuberculous ulcers in the small intestines, and tuberculous peritonitis. In the white substance of the right cerebellar hemisphere there was a globular caseated tubercle destroying the dentate nucleus on the right side, pushing the cerebellar vermis to the left and reaching the dorsal surface of the cerebellum (see Fig. 1). A definite internal hydrocephalus was present. The tubercle showed no evidence of progression but rather a stage of involution.

This case does not need any comment and shows plainly an instance where the diagnosis, "Pseudo-Ménière's disease," was erroneous.

DeKleyn and Versteegh distinguish between four kinds of Ménière's attacks: *1.* patients who suffer from dizzy spells independent of position of head; *2.* patients who suffer from vertigo and nystagmus with the head in certain positions in space ("positional nystagmus") *3.* those who have symptoms with the head in certain positions in relation to the body; *4.* those who have vertigo, which, however, is not character-

ized by the feeling of whirling of their own body, but which is rather characterized by the fact that the patients perceive the surroundings in a slanting position. I do not agree with this division, since it adds new problems and renders the analysis more difficult. Following our definition, we diagnose Ménière's attacks only in those cases which are described by DeKleyn and Versteegh under the first heading, if these dizzy spells are combined with cochlear symptoms.

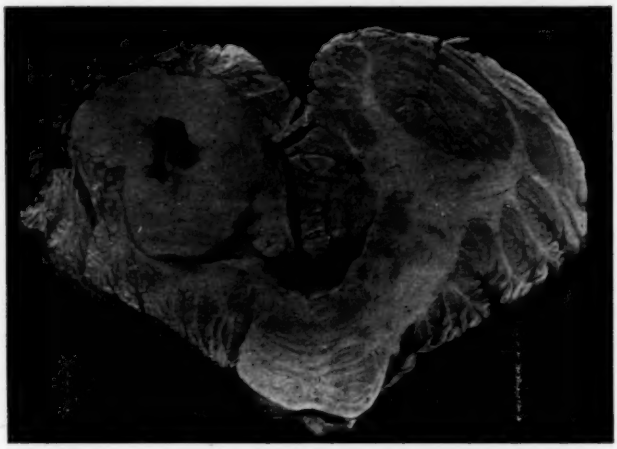

Fig. 1.

ETIOLOGY.

There are many diseases which may or may not be accompanied by Ménière's syndrome. Among these diseases we mention the following: leukemia (Alexander, Schwabach), head injuries, arteriosclerosis of the brain (Manasse), arteriosclerosis of the vertebral and internal auditory artery (Dandy, Olivecrona,[52] Hofer[35]), neurofibroma of cochlear nerve (Wittmaack[64]), cerebellar tumors, Paget's disease of the temporal bone (Brunner,[8] Brunner and Grabscheid[14]), encephalitis (Videbech[62]), polyneuritis cerebralis menieriformis, with or without herpes zoster oticus (v. Frankl-Hochwart), tabes, syphilitic neurolabyrinthitis, polyneuritis (Berggren[4]), helminthiasis, sunstroke (Guettich[31]), chronic adhe-

sive processes of middle ear (Brunner, Mygind and Dederding[48]), otosclerosis. These represent an incomplete list of all diseases which may be accompanied by Ménière's syndrome.

In all these conditions, however, the Ménière's syndrome, if it occurs, is a symptom of a certain well known disease. Consequently, we speak of a symptomatic Ménière's syndrome, an expression which means that in these circumstances Ménière's syndrome is one of the symptoms of a certain disease. The symptomatic Ménière's syndrome is not supposed to be discussed in this paper. However, in the section, "Pathology," it will be necessary to return to this form of Ménière's syndrome.

Besides these cases with symptomatic Ménière's syndrome, there is quite a large group of cases in which no etiology can be found. These have been denoted as "Ménière's disease" and not as "Ménière's syndrome." Dandy states that we know of no cause for "Ménière's disease," which is supposed to be "merely an explosion of the function of the inner ear." Also, Mygind and Dederding[50] define "Ménière's disease" as an ear disease with periodically varying acoustic and labyrinthine symptoms, which do not present any special etiology. Munro[47] shares the same view. I do not speak of "Ménière's disease" in these cases, but rather of an idiopathic Ménière's syndrome, since it is very difficult to think of a disease which has no etiology and no pathology and which is "merely an explosion of the function of the inner ear." We must rather assume that there is a definite cause for the idiopathic Ménière's syndrome. Unfortunately, that cause is not exactly known at the present time. But there is hope that it will some day be found. When this occurs, no idiopathic Ménière's syndrome will exist, since this syndrome will be a symptom and not a morbid entity under all circumstances.

Idiopathic Ménière's syndrome has been studied intensively in recent years. At first the metabolism was studied in these cases. Fairen[25] found that the creatin and the creatinin in the blood was increased. Furstenberg, *et al.*,[27] state that the symptoms of Ménière's syndrome are due to the retention of sodium by the body. If a sufficient supply of water is available it will likewise be retained, but this fact is not of primary importance. Mygind and Dederding[48] object that Furstenberg did not stress the importance of the examination of

the auditory conditions, this being the only indicator of the adequacy or inadequacy of treatment.

Contrary to Furstenberg, *et al.*, Mygind and Dederding believe that the essential factor in idiopathic Ménière's syndrome is a propensity to water retention generally as well as locally, but above all in the ears. These disturbances in the water metabolism have a tendency to periodic fluctuation, due to a series of external and internal factors which seem to exert their action essentially through a partly local, partly general vasomotor and capillarimotor-dysfunction.

It is obvious that the examination of the metabolism has not yielded definite results. Consequently, other findings were associated with the idiopathic Ménière's syndrome, such as allergy and focal infection. In 1923, Duke[23] described five cases of Ménière's syndrome caused by allergy; later, Dean, Agar and Linton[22] and, finally, Jones[37] again called attention to the fact that the phenomenon of allergy does affect the Eustachian tube, the middle and the internal ear, even though scientific proof is lacking. Also, Granstroem and Nylen[30] stress the importance of allergy in the etiology of Ménière's syndrome. On the other hand, Thornvall and Crowe[18] could not find allergy as cause of this syndrome.

As far as focal infection is concerned, Wright[67] points out that cases of labyrinthine vertigo, which have been regarded as of unknown or doubtful etiology (*viz.*, idiopathic Ménière's syndrome), can all be grouped together as belonging to a single disease which he calls "focal labyrinthitis." That this labyrinthitis is secondary to a focus of infection is shown by the invariable presence of such a focus and, to a greater extent, by the arrest or even cure of the disease when such focus is eradicated.

Prior to Wright, G. Shambaugh,[59] in 1917, and G. Mackenzie,[43], in 1929, proposed the same idea. Even though in some cases focal infection is of some importance, Wright seems to be too certain. We should not forget that even today many physicians consider the teaching of focal infection not only as a science but also somewhat of a religion. Second, Dandy and Crowe definitely refuse to include focal infection as a possible etiology in idiopathic Ménière's syndrome. Third, we will see later that even the therapeutic results of Wright him-

self do not permit such definite conclusions; however, I wish to repeat that I do believe focal infection to be one of the causes of idiopathic Ménière's syndrome and that I saw cases with focal infections originating in the gall bladder. I object very strongly, however, to calling the idiopathic Ménière's syndrome a "focal labyrinthitis."

Matzdorf[45] believes there is a connection between hemicrania and Ménière's syndrome. Brunner and Spiegel,[13] in 1921, described such cases as "hemicrania otica," but these cases are rather rare. Just believes that Ménière's syndrome is an angioneurosis of the acoustic nerve due to an irritation of the mucous membrane in the superior nasal meatus. Dandy, McKenzie and Olivecrona[52] found loops of arteries pressing on the VIIIth nerve as a possible cause of the symptoms of Ménière's syndrome.

Summarizing, we see that there is not much definite knowledge concerning the etiology of idiopathic Ménière's syndrome. And even the findings which are considered important by each author are not confirmed by others. Very often the observation of the cases is not sufficiently accurate nor extended over an adequate period of time. A great amount of work is yet to be accomplished before we get a clear view of the etiology.

PATHOLOGY.

Although the symptoms of the original case of Ménière were due to a hemorrhage into the inner ear, the modern conception of idiopathic Ménière's syndrome cannot be explained by hemorrhage into the inner ear. This, however, may sometimes occur in symptomatic Ménière's syndrome.

Since patients always survive the idiopathic Ménière's syndrome, there is rarely an opportunity for postmortem examinations in these cases. Consequently, the hypotheses grew up like mushrooms after the rain. The origin of the syndrome was localized in the brain (Thornvall), in the VIIIth nerve (Dandy), in the internal ear and in the middle ear. As a matter of fact, there is no further choice.

Brain: Thornvall's view is a pure theory, like the explosion theory of Dandy. He believes that the nerve cells in the otolithic centre of the brain stem are gradually charged with

irritants and when the tension has reached a certain point, a sudden discharge occurs, which is the Ménière's attack. This theory may or may not be believed; at any rate, it is very difficult to prove. This is more difficult when one considers that a symptomatic Ménière's syndrome is a rare occurrence in organic brain diseases. We know very well that every textbook teaches the occurrence of Ménière's attacks in disseminated sclerosis, encephalitis, etc., and the case of Videbech is supposed to prove such an occurrence, with the aid of an autopsy. In our experience that statement does not hold true. Unquestionably there occur dizzy spells and other spells in the mentioned brain diseases. These, however, are not Ménière's syndrome, if one accepts the definition as given above. The importance of making an exact diagnosis is shown by Videbech's case, in which encephalitis was found. However, it is not clear that the patient actually had Ménière's attacks. The author speaks of "crises syncopal" and "crises d'evanouissement," combined with tinnitus and nausea, but no mention is made of vertigo of labyrinthine origin, particularly since the patient had no spontaneous nystagmus. Furthermore, the examination on the turning chair revealed a very definite hypoexcitability of the labyrinths, especially on the left side, while the labyrinths responded normally to the caloric test. This finding is very unusual in symptomatic or idiopathic Ménière's syndrome. Consequently, the case of Videbech does not prove definitely the occurrence of symptomatic Ménière's syndrome in encephalitis.

I am, of course, not able to state that in organic brain disease a typical Ménière's syndrome, as defined above, does not exist, but I do believe that this occurrence is not frequent. Consequently, we must say that if the symptomatic syndrome occurs infrequently in organic brain diseases, it is most unlikely that the idiopathic Ménière's syndrome may develop without active assistance of the ear and its nerves. This opinion is in agreement with the opinion of Crowe and is proved by the case of Berggren, who examined the brain in a case of idiopathic Ménière's syndrome. There were no pathological changes in the brain stem except for a degeneration of the nuclei of Gol and Burdach, a finding which has nothing to do with Ménière's syndrome.

Acoustic Nerve: As already mentioned, Dandy localizes the idiopathic Ménière's syndrome in the acoustic nerve; however,

the arguments promulgated by Dandy are not satisfactory. He states that a lesion of the inner ear could not affect both the cochlear and labyrinthine divisions. This statement is not in agreement with experiences of otologists. It is a clinical rule that acute symptoms of irritation or destruction of both divisions of the inner ear indicate a disease within the inner ear, while acute symptoms of irritation or destruction of one division of the inner ear indicate a disease behind the inner ear, *viz.*, in the nerve. Consequently, the occurrence of both, labyrinthine and cochlear symptoms in Ménière's syndrome, particularly in Ménière's attack, points to the inner ear as the site of the disease and not to the nerve. The well known observation that during Ménière's attacks, as well as between the attacks, the dizziness is dependent on movements of head is further proof in the same direction. Moreover, McKenzie, Furstenberg, Crowe, Hallpike and Cairns examined microscopically pieces of the vestibular nerve in cases of Ménière's syndrome without finding any pathologic change. Only André-Thomas, Aubry and Ombrédanne found in one case an increase of connective tissue within the nerve.

Although there is not much proof that the idiopathic Ménière's syndrome originates within the VIIIth nerve, a symptomatic Ménière's syndrome may occur in diseases of the VIIIth nerve as in small acoustic tumors, or in polyneuritis cerebralis ménièriformis.

Internal Ear: As far as the inner ear is concerned, the case of Wittmaack is interesting. The patient was not thoroughly examined but the diagnosis of "Ménière's disease" was made. The only pathologic change Wittmaack found at autopsy was an obstruction of the cochlear aqueduct due to a squeezed concretion. He draws the conclusion that this concretion hindered the outflow of the perilymph, thus producing an increase of pressure within the perilymphatic spaces of the inner ear and, consequently, an attack of dizziness. Since Wittmaack found the same concretions obstructing the cochlear aqueduct also in another case of Ménière's syndrome (which had also a neurofibroma of the cochlear nerve), he compares the Ménière's attacks with the crises due to stones in the gall bladder or in the kidney.

On the other hand, there was no pathology in the temporal bones or in the brain in the case of Berggren, although

the patient, a woman, age 74 years, had severe Ménière's attacks and a progressive deafness on the left side. No statement is made as to whether or not particularly the cochlear aqueduct was examined in that case. We omit the case of Videbech since we do not know whether the patient had actual Ménière's attacks.

The findings of Hallpike and Cairns are important in considering the pathology of idiopathic Ménière's syndrome. They describe two cases:

Case 1: A man, age 63 years, complained of vertigo for three years. The caloric test did not give a response on the left side but did on the right. The left vestibular nerve and part of the left cochlear nerve were divided. Three days later, the patient died, and both temporal bones were examined microscopically. On both sides there was a chronic adhesive process of the middle ear. In the left temporal bone there was a gross dilatation (ectasia) of the scala media of the cochlea and of the saccule, with obliteration of the perilymph spaces of the vestibules and the scala vestibuli. The left aqueduct of the cochlea was filled throughout its length with a pinkish coagulum; the right aqueduct was patent. Finally, there was a degeneration of Corti's organ in the left temporal bone.

Case 2: A man, age 28 years, who suffered from attacks of vertigo for four years. He heard a watch at 6 inches in the left ear and 4 feet in the right. Air conduction was greater than bone conduction. Cold caloric tests produced nystagmus and moderate vertigo on both sides. The left vestibular nerve was divided. Two days later, the patient died. The left temporal bone was examined and there was found: 1. gross dilatation (ectasia) of the saccule and the scala media of the cochlea, with obliteration of the perilymphatic cistern and the scala vestibuli, the aqueduct of the cochlea being occupied throughout its length by dense masses of red cells with a few scattered small lymphocytes and polymorphonuclear leukocytes; 2. degeneration of Corti's organ; 3. degeneration of the epithelium of the maculae and of the cristae of the semicircular canals; 4. degeneration of the stria vascularis.

Hallpike and Cairns identify their microscopic findings with the hydrops labyrinthi described by Wittmaack as representing the reaction of the inner ear to damage from bacterial or other toxins reaching it through the round window or via the blood stream. With this statement the authors are in contradiction to Wittmaack, who definitely states that the hydrops labyrinthi cannot be considered as a cause of Ménière's attacks. Furthermore, Hallpike and Cairns believe that the attacks are due to rapidly initiated bouts of asphyxia of the labyrinthine end-organs brought about by extremely rapid rises of fluid pressure in response to relatively small volume increase in the endolymph.

One cannot pretend that the examinations of Wittmaack, Berggren, Hallpike and Cairns contribute a definite knowl-

edge concerning the pathology of idiopathic Ménière's syndrome. The number of thoroughly examined cases is too small. We found no pathology (Berggren), obstruction of the cochlear aqueduct with consequent increase of pressure in the perilymph spaces (Wittmaack), and hydrops labyrinthi with consequent increase of pressure in the endolymph spaces (Hallpike and Cairns). Nevertheless, all these findings point to the fluids in the inner ear as the source of the disorders producing Ménière's syndrome. Consequently, the physiology of the fluids has to be considered. Unfortunately, that question raises new problems, since there are many gaps in our knowledge. The usual concept is as follows: the endolymph is an actual secretion produced in the stria vascularis and, according to Wittmaack, in the organ of Corti and the maculae. The outflow of endolymph is accomplished: *1.* by diffusion into the perilymph space, particularly through Reisner's membrane; *2.* by little channels in the saccus endolymphaticus; and *3.* according to Guild, by the epithelium of the saccus endolymphaticus. The perilymph is not cerebrospinal fluid, but a dialysate produced by diffusion from the blood vessels covering the walls of the perilymph spaces. The outflow is accomplished: *1.* by the cochlear aqueduct; *2.* by the veins covering particularly the walls of the scala tympani and emptying into the posterior spiral vein.

In order to obtain a better understanding of the pathology of the labyrinthine fluids, the pathology of hydrops labyrinthi (Wittmaack) and otitis interna vasomotoria (Brunner) must be considered. After injuring the skull of a guinea pig without producing a fracture, the microscopic examination of the temporal bones reveals a normal middle ear, but the inner ear shows perivascular infiltrations with lymphocytes, a serous exudate in the perilymph spaces containing a few lymphocytes, and a moderate dilatation of the scala media. Since these changes cannot be considered artificial, the question arises as to the manner of classifying these pathologic findings. It cannot be called hydrops labyrinthi. According to Wittmaack, the hydrops labyrinthi is due to an invasion of toxic, chemical or even physical irritants passing the round window or the internal meatus into the inner ear. Nothing like that happened in our experiments. Second, the hydrops labyrinthi is produced by a hypersecretion of the stria vascularis. In our experiments the changes originated apparently

from the blood vessels. Third, the hydrops consists of a hyperproduction of normal endolymph. In our experiments there was a definite exudate and lymphocytes in the perilymph spaces, in addition to a hypersecretion of endolymph. Fourth, in hydrops the changes are found in the endolymph spaces. In our experiments the most marked changes were found in the perilymphatic spaces and less in the endolymphatic spaces.

Since the pathologic finding is not a hydrops labyrinthi, it can only be an otitis interna serosa, such as is familiar to every otologist. This internal serous otitis, however, showed some peculiarities. Usually the internal serous otitis is due to an invasion of bacteria or, more frequently, of bacterial metabolites into the inner ear, the source of the bacteria being the middle ear, the meninges or (rarely) the blood stream. In our experiments the middle ears and meninges are normal and no blood stream infection was present. Consequently, it is not an otitis interna serosa of bacterial origin, but a special form of otitis interna serosa. It is not difficult to explain the pathogenesis. Since vasomotor disturbances in the brain are one of the chief consequences of an injury of the skull, and since the blood vessels of the inner ear belong to the circulus arteriosus Willisii, it is logical to explain all changes found in the inner ear of the guinea pigs as being produced by vasomotor disturbances. Consequently, we call the serous internal otitis found in our guinea pigs, otitis interna vasomotoria. This is just an ordinary serous internal otitis produced by any irritant affecting the vasomotor nerves of the inner ear, except bacterial irritants.

Unfortunately, the otitis interna vasomotoria has not been studied as well as hydrops labyrinthi, since my observations until now were neither controlled nor continued. We may, however, suppose that the otitis interna vasomotoria, like the hydrops labyrinthi, never produces purulent exudate and that a mild degree of the disease may disappear without any after-effects. On the other hand, a severe degree of the disease may produce severe changes in the inner ear, such as degeneration of the end-organs and dilatation of the pars inferior of the inner ear, inasmuch as severe degrees of otitis interna vasomotoria always are combined with a concomitant hydrops labyrinthi.

These experimental findings can be confirmed by findings in human temporal bones. In 1921, I examined the following

case: A man, P. J., age 58 years, became suddenly deaf nine years previously after a common cold. Since that time he often had attacks of dizziness, which were accompanied by projectile vomiting and headache. On July 9, 1921, we found a chronic middle ear catarrh on both sides, and the labyrinths were slightly hyperexcitable. Twenty days later the following ear findings were noted: Chronic middle ear catarrh present on both sides. The patient was stone-deaf on the left side, and on the right he heard words if shouted into the ear. There was no spontaneous nystagmus, and a hypoexcitability of marked degree in both labyrinths was present. Two and one-half months later the patient died of an erysipelas.

The microscopic examination revealed a typical Paget's disease of the temporal bones. There was no inflammation in the middle ear. In the inner ear, on the left side, there was a very marked ectasia of the scala media, saccule and partially of the utricle, a marked degeneration of Corti's organ and the cochlear nerve, an atrophy of the maculae and cristae, and an obliteration of the cochlear aqueduct. In short, there were the same findings as in the cases described by Hallpike and Cairns, except Paget's disease of the bone. The inner ear on the right side showed the same changes, although the dilatation of the pars inferior was missing and the cochlear acqueduct was patent.

It was not possible at that time to give a definite explanation for the findings in that case. It was striking that the dilatation of the pars inferior was present on that side only where the cochlear aqueduct was found to be obliterated.

A much better understanding was obtained when I examined, in 1931, the following case: A man, M. D., age 65 years, who had diminished hearing in the last few years but had neither dizziness nor headache. He was previously addicted to drink. Death resulted from an apoplexia cerebri. The microscopic examination revealed a typical Paget's disease of the temporal bones. There was no inflammation in the middle ears, but there was a typical serous internal otitis on both sides, and both cochlear aqueducts were patent. The organ of Corti was partially degenerated, but a definite degeneration of the maculae and cristae were not present.

Among five cases of Paget's disease I found serous internal otitis four times; in one case it was missing. All these obser-

vations lead to the following conclusions, providing that our findings will be confirmed by other investigators: If an osteitis, such as Paget's disease, reaches the endosteum of the inner ear to a large extent, a serous labyrinthitis may develop. Since there are no pyogenic bacteria in the middle ear, meninges or in the blood stream, and since Paget's disease is not of bacterial origin according to our present knowledge, the serous internal otitis found in our cases is not produced by bacteria or their metabolites. Consequently, it is an otitis interna vasomotoria, as has been found also after experimental head injuries.

As in every serous labyrinthitis, the otitis interna vasomotoria may disappear without damaging the end-organs of the inner ear, the exudate leaving the inner ear particularly through the cochlear aqueduct. If the patient dies in that stage of the disease, no pathologic change can be discovered in the inner ear.

The otitis interna vasomotoria, however, may return, providing that the pathogenic process which affects the vasomotor nerves, *e.g.*, Paget's osteitis, remains within the body of the patient. If that is the case, or if the otitis interna vasomotoria becomes severe, the end-organs must be damaged; at first in the pars inferior, *viz.*, Corti's organ and macula sacculi; later in the pars superior, *viz.*, macula utriculi and cristae of ampullae.

Since the exudate, as already mentioned, leaves the inner ear particularly through the cochlear aqueduct, a stagnation of that exudate within the narrow canal easily occurs, with resulting obstruction of the canal by exudate, connective tissue or concretion. Consequently, the obstruction of the cochlear aqueduct is not, as Wittmaack believes, the cause; it is rather the consequence of the otitis interna vasomotoria (*viz.*, of Ménière's attack).

If the chief way for the outflow of the exudate and the perilymph, namely, the cochlear aqueduct, is cut off, the exudate stays in the perilymph spaces and passes finally by diffusion into the endolymphatic spaces. Consequently, the membranes of the inner ear, particularly Reisner's membrane, remain under entirely abnormal physical conditions, *i.e.*, the exudate in the perilymph spaces attracts the membranes by viscosity into the perilymphatic space, and the concomitant

hydrops labyrinthi pushes them in the same direction. This displacement of the membranes increases and may become permanent when the exudate in the perilymphatic spaces is organized into connective tissue. This may happen in serous labyrinthitis, as we know, in a relatively short time. It is obvious that this displacement can occur only in those parts of the inner ear where perilymphatic tissue is missing under normal circumstances, *viz.*, in the cochlea, the saccule and partly in the utricle. Since the organization of the exudate requires a certain time, we find the dilatation (ectasia) of the pars inferior of the inner ear only in cases who have suffered from Ménière's syndrome for a long period of time, as our case P. H. and the cases of Hallpike and Cairns.

From the above the various findings in the inner ear in Paget's disease of the temporal bones can be explained. The next question which arises is whether or not the described pathologic changes in the inner ear are the actual cause of Ménière's syndrome. The following case may answer that question:

This was a woman, K. B., age 68 years, who became suddenly ill with an attack of whirling, dizziness, projectile vomiting, tinnitus and diminution of hearing on both sides. The Wassermann in the spinal fluid was positive. A functional test of the ears was not performed. Two and one-half weeks after the onset of Ménière's attack the patient died.

The microscopic examination revealed a typical Paget's disease of the temporal bones. There was no inflammation of the middle ear but there was a typical serous internal otitis with a definite degeneration of all end-organs in the inner ear.

I believe that this case "closes the circuit," since it shows clinically a typical Ménière's attack a short time before death and, pathologically, a typical otitis interna vasomotoria. Consequently, we feel justified in stating that the Ménière's attack corresponds to an attack of otitis interna vasomotoria. The Ménière's attacks differ in degree, as do also the attacks of otitis interna vasomotoria. Between the attacks the functional testing of the ears may yield different results, depending on the permanent changes of the inner ear produced by the otitis interna vasomotoria and depending on the condition of the middle ear.

The analogy between otitis interna vasomotoria and rhinitis vasomotoria is obvious. There are, however, some impor-

tant differences: *a.* The watery secretion in rhinitis vasomotoria originates from the blood vessels and the glands of the nasal mucous membrane. In the inner ear there are no glands. *b.* The blood vessels of the nasal mucous membrane are embedded in soft tissue. Consequently, a marked swelling or shrinking of the mucous membrane can occur. The blood vessels of the inner ear lie either in narrow, bony channels or just below the endosteum. A marked swelling of these tissues is not possible. *c.* The secretion produced by the rhinitis vasomotoria easily leaves the nose either through the nostrils or through the choanae. The secretion produced by the otitis interna vasomotoria can leave the inner ear by very narrow channels only. Consequently, it may very easily be retained within the inner ear. *d.* In the nose there are no structures which could be damaged by the secretion. In the inner ear there are the delicate end-organs, which may be damaged temporarily as well as permanently by the secretion.

It is true that our conception of the pathology is based upon the study of the symptomatic Ménière's syndrome (head injuries, Paget's disease). However, since in our opinion the idiopathic Ménière's syndrome is just a symptomatic Ménière's syndrome with an unrecognizable etiology, it seems justifiable to assume that the pathology in both syndromes is alike, as will be proved by the cases of Wittmaack, Berggren, Hallpike and Cairns, and possibly by the case of Videbech.

Middle Ear: There are two nonpurulent diseases of the middle ear which sometimes are accompanied by a symptomatic Ménière's syndrome; *viz.*, otosclerosis and chronic adhesive processes of the middle ear. In neither of these diseases have pathological findings of the inner ear, explaining the Ménière's attacks, been obtainable. Consequently, nothing is known for sure; however, it is possible to conjecture as to the pathologic findings. As far as the otosclerosis is concerned, Ménière's attacks can be explained by localization of an otosclerotic focus in the concavity of the superior semicircular canal or by localization of an otosclerotic focus in the wall of the internal meatus of the temporal bone or by a combination of otosclerosis with a chronic adhesive process of the middle ear.

Despite contrary reports, I must insist that chronic adhesive processes of the middle ear are not infrequently com-

bined with symptomatic Ménière's attacks. Since I called attention to these conditions 16 years ago, I saw a great number of similar cases. The pathologic explanation is hypothetical up to now. It must be remembered that in chronic adhesive processes there is always some exudate between the adhesions, containing sometimes a large number of cells, and, furthermore, that this exudate is most often situated within the niches of the windows. It is quite possible to suppose the proteins originating from that exudate may pass by diffusion through the windows, thus producing an otitis interna vasomotoria. That hypothesis is even more acceptable if one considers the clinical observation that in chronic adhesive processes the vasomotor nerves of the ear often seem to be hyperexcitable, as may easily be seen on the blood vessels of the drum and the external auditory canal, which very rapidly get hyperemic even after a short and gentle examination with the otoscope.

There is no reason for considering the middle ear as the origin of the idiopathic Ménière's syndrome.

PROGNOSIS.

If one discusses the prognosis of Ménière's syndrome he must have in mind three symptoms in which the patient and the attending physician are interested; *viz.*, dizziness, progressive deafness and tinnitus. The gradual decrease of labyrinthine excitability is interesting for the physician but less for the patient.

The dizziness is most marked during the spells, less between the spells. v. Frankl-Hochwart, who reported 208 cases of Ménière's syndrome, stated that the prognosis, as far as the dizziness is concerned, is relatively good, although it may persist from a few weeks up to a few years. The recovery sometimes occurs in such a manner that there is a sudden cessation of the attacks of dizziness; more frequently the attacks decrease gradually in intensity and frequency. The cases of v. Frankl-Hochwart are not incontestable, since the author worked at a time when there was much confusion about "Ménière's disease," and when the modern labyrinthine tests were not as yet discovered. The number of cases, however, observed by v. Frankl-Hochwart is so great that there

were undoubtedly cases of idiopathic Ménière's syndrome among them.

Moreover, Thornvall expresses the same opinion as far as the prognosis of the dizziness is concerned. He observed 15 patients for a long period of time and found that the dizzy spells lasted from six months to 12 years and ceased afterwards, although only symptomatic treatment or no treatment had been administered. My experience agrees with the reports of v. Frankl-Hochwart and Thornvall. I only wish to add that I have observed cases in which the dizzy spells disappeared, but not the spells of spontaneous nystagmus. These patients noticed headache and a general uncertainty of their body balance, but they could rise from bed without help. They could even walk without help, providing their attention was fixed more than normally. In examining these patients it was a surprise to find a spontaneous nystagmus of third degree to one side. Crowe does not report spontaneous cessation of the dizzy spells, but he does report (in agreement with v. Frankl-Hochwart) spontaneous remissions. Of 117 of Crowe's patients, 48 had intervals between the attacks that varied from one to two months up to 12 years.

Summarizing, there can be no doubt that a spontaneous cessation of the dizzy spells in Ménière's syndrome is possible. It is even likely, although it cannot be foretold in every case, if and when that will happen. This experience fits into the physiology of the human labyrinth very well. Already, Maupetit has shown that there exists a habituation to labyrinthine dizziness, and every case of acute purulent labyrinthitis proves that fact, when one remembers that the dizziness may disappear, although a spontaneous nystagmus of second degree persists. Consequently, it is hard to understand what makes Dandy say, "Once Ménière's disease begins, it never ends spontaneously." As far as the dizziness is concerned, that statement certainly does not hold true. I only wish to quote McKenzie, Olivecrona and Horrax as neurosurgeons who mention cases of Ménière's syndrome who lost their dizzy spells spontaneously or after conservative treatment.

The prognosis as far as the hearing is concerned is worse, as stated already by v. Frankl-Hochwart. If the symptom-complex is of but short duration it is quite possible for the Ménière's syndrome not to affect the hearing at all. If, how-

ever, an impairment of hearing on one side sets in, then it is, as a rule, progressive and may affect the other side, too, although complete bilateral deafness is not very frequently met.

It is interesting that all observers (v. Frankl-Hochwart, Thornvall, Crowe and others) report cases in which after a Ménière's attack the hearing was markedly improved and remained that way. Thornvall points out that such an inexplicable, permanent improvement of hearing after an attack usually occurs at the commencement of Ménière's syndrome. Unfortunately, one but seldom meets such cases.

The worst prognosis is attached to the tinnitus. There is scarcely a case of Ménière's syndrome in which this symptom was not present if observed during a sufficiently long period of time. On the other hand, Ménière's syndrome very often sets in with tinnitus, and that tinnitus may persist, even when the patient is completely deaf. By far more rare are those cases in which the tinnitus ceases prior to the complete loss of hearing.

There is a great difference between the tinnitus and vertigo, since habituation to tinnitus seldom occurs. A further difference is the fact that the disappearance of vertigo is much more dependent on the inner ear than that of tinnitus. If the labyrinth is destroyed, the vertigo usually ceases, providing there is no disease in the nerve or in the brain which produces vertigo of its own. On the contrary, the tinnitus may persist if the inner ear is destroyed, a fact which was already striking in the past, when the indications for labyrinth operations were more liberal than they are today. While a patient may become accustomed to dizziness by a sort of central compensation, no difference existing whether or not the causal disease of the inner ear persists, the patients often suffer from continued tinnitus, even when the causal disease of the inner ear is removed. A central compensation for tinnitus is not known. Consequently, the vertigo has not only a better prognosis than the tinnitus, since a central compensation for dizziness and even for nystagmus is well known, but it can also be more easily influenced by treatment. In order to get a sure judgment concerning the effectiveness of treatment in Ménière's syndrome, the question whether or not the tinnitus was influenced by the treatment must be answered.

TREATMENT.

The treatment of the symptomatic Ménière's syndrome depends on the causal disease, if there is a proper treatment available for that causal disease as, for instance, in lues or in chronic adhesive processes of the middle ear. If there is no adequate treatment for the causal disease, as, for instance, in Paget's disease or after concussion of the brain, the symptomatic Ménière's syndrome has to be considered as an idiopathic Ménière's syndrome.

As far as the treatment of the idiopathic Ménière's syndrome is concerned, there exists a radical and a conservative treatment. The radical treatment, which originated with Charcot, consists of a destruction of the affected ear. This can be accomplished by different methods: *a.* by administration of certain drugs, such as chinin (Charcot); *b.* by surgical opening of the labyrinth and curetting its contents (Hautant, Putnam); and *c.* by division of the VIIIth nerve (Dandy). There have been recommended other surgical procedures, such as the opening of the saccus endolymphaticus (Portmann), operations on the cervical part of the sympathetic nerve (Brunetti) or injection of alcohol either through a hole in the lateral semicircular canal (Mollison) or through the oval window (Peacock). These procedures, however, have not become popular.

The conservative treatment is still in the experimental stage. Besides the usual hygienic measures (dust-free air, no excitement of any kind), the administration of bromides, luminal, pilocarpin (recently rediscovered by Leichsenring), atropin, adrenalin, ovarian preparations, iron, radiation with carbon arc light, carbonic acid baths, sulphur baths, pine needle baths, natural sun baths and salt brush baths have been recommended. Thornvall succeeded with parathyroid tablets and calcium. Some otologists recommend spinal puncture, which I have combined with inflation of air into the spinal canal. I have recommended the combined treatment with iodin and calcium (atrocal or afenil). v. Frankl-Hochwart had good results with galvanization of the head, and German with ultra short waves.

It is obvious that this conservative treatment consists of a groping in the dark, although unquestionably quite a number of cases were treated successfully. It was indicative of great

progress that Mygind and Dederding on the one side, and Furstenberg, *et al.*, on the other side undertook the task of putting the conservative treatment on a scientific basis. According to their research work, the cases were treated with dietary measures, the diet consisting essentially of a restriction of the intake of fluids and sodium, and a prevention of accumulation of sodium by the body by means of administration of acid-producing salts, such as ammonium chloride. Furstenberg describes in great detail the foodstuffs which should be allowed and those which should be restricted. Brown reports good results with this treatment. In the conservative treatment we include the removal of all focal infection in the body, as recently re-emphasized by Wright.

It is obvious that there are quite a number of therapeutic measures employed in Ménière's syndrome. Everybody is successful with his own treatment, since nearly everybody has his own ideas about Ménière's syndrome. Progress never originates from confusion; therefore, we repeat that we always have in mind the idiopathic Ménière's syndrome, and we insist that in every case it must be decided at first if one is dealing with a symptomatic or idiopathic Ménière's syndrome. Only if the employment of all clinical methods fail to reveal an etiology may the case be called one of idiopathic Ménière's syndrome. Furthermore, the question must be answered, whether or not the case is to be treated by surgical or conservative measures. In order to do this one has to know the results of the surgical and conservative methods.

Unfortunately, the majority of reports simply speak of Ménière's syndrome. Consequently, if one wishes to learn the therapeutic results in Ménière's syndrome he has to limit himself to statistics covering a large number of cases, assuming that among these cases there will be a sufficient number of cases of idiopathic Ménière's syndrome which have been observed for an adequate period of time.

As far as radical treatment is concerned, exact records are only available concerning the division of the VIIIth nerve. Crowe reports 49 cases in which both divisions of the VIIIth nerve were divided. In all cases the dizziness ceased, but only in 24 cases (49 per cent) did the tinnitus disappear, and all cases were deaf on the operated side. Furthermore, Crowe reports 72 cases in which the vestibular nerve only was divided. Among these cases, in 22 instances (30.5 per cent)

the deafness in the affected ear grew worse; in 36 (50 per cent) the deafness in the affected ear remained unchanged, and in 14, or 19.5 per cent (Crowe writes apparently by mistake, 22.2 per cent), the deafness on the affected side was much improved, while the dizziness apparently in all cases disappeared. All observers (Dandy, Crowe, McKenzie, Coleman, Olivecrona, Hallpike and Cairns, and Horrax) agree that the tinnitus disappears only in a certain percentage of cases, even if the entire VIIIth nerve is divided; however, it was not possible to obtain exact figures.

As far as conservative treatment is concerned, there are at our disposal only the results published by Mygind and Dederding, and Wright. Mygind and Dederding have treated 157 patients with their dietary measures. One hundred fifty of these patients have recovered from giddiness and nystagmus, and in 85 the deafness was distinctly improved on dismissal from the hospital. The results have been permanent in 67 patients for giddiness, and in 29 patients as far as the deafness was concerned.

Wright has eradicated all foci of infections he could reach in 23 cases. The results have been as follows: 10 cases were cured; *viz.*, the dizziness disappeared and the hearing returned to the normal. In 13 cases the dizziness disappeared but the hearing remained unaltered, or the improvement was not definite. It is regrettable that all these statistics do not stress the proper importance of the tinnitus since, according to the prognosis of Ménière's syndrome, the tinnitus is the most obstinate symptom and shows the least propensity for spontaneous remissions and for spontaneous cessation. Consequently, that symptom is the best criterion of the success or failure of treatment. One must further emphasize the failure of the authors to signify the period of observation of their patients. Only Crowe mentions that the average time elapsed between the operation and the last examination was 2.2 years. Nevertheless, we have to compare the present statistics since none better are available. The comparison of these figures yields the following table:

Author	Number of Cases	Definite Cure of Dizziness	Definite Improvement of Deafness
Crowe	72	100.0%	19.5%
Mygind-Dederding	157	42.7%	18.5%
Wright	23	100.0%	43.5%

As far as the definite cure of dizziness is concerned, Dandy and Crowe had the same results with division of the VIIIth nerve as Wright had with the eradication of foci of infection. Mygind and Dederding's results were nearly 50 per cent worse. The last number, however, needs an explanation. Mygind and Dederding divide their results into transitory and permanent cures. If we consider the transitory results, they cured, as far as dizziness is concerned not 42.7 per cent but 95.5 per cent of their cases. Since it is not certain that Wright, Dandy and Crowe observed their cases for the same period of time that Mygind and Dederding did, it cannot be definitely said that the smaller number of cures reported by Mygind and Dederding must be explained by the lesser effectiveness of their treatment. At any rate, the table proves that it is possible to obtain with conservative measures, particularly with eradication of foci, almost a similar percentage of cures as with the division of the nerve.

As far as definite improvement of the hearing is concerned, there is not much difference between the number of cures obtained by radical and the number obtained by conservative treatment. Here, the results of Wright are far better than those of Dandy and Crowe. It has, however, to be remembered that the number of cases observed by Wright is far smaller than those observed by Dandy and Crowe. In addition, it is possible that the cases which had to be treated by Dandy and Crowe were more advanced than those of Wright.

Summarizing, we must say that the results by the division of the VIIIth nerve are not convincingly superior to those obtained by more conservative methods, particularly if we keep in mind, first, that there exists unquestionably a spontaneous cessation or remission of vertigo for a long period of time; and second, that the results of the operation as far as tinnitus is concerned are definitely worse than those for dizziness. In addition, we cannot entirely agree with Dandy when he says: "The treatment entails practically no risk." The two cases, published by Hallpike and Cairns, who died after the division of the nerve prove the contrary. Also, McKenzie lost one case among 12, and, finally, Rutherford reports one fatal case. We, of course, admit that these fatalities rarely occur, since Dandy's last report states that there was no fatality among 264 operations. These brilliant statis-

tics, however, do not exclude the fact that other surgeons did observe fatalities and that this is noteworthy in a disease which never leads to a fatality of itself.

But there are lesser risks which have to be considered. Dandy himself saw postoperative facial paresis in two instances, and so did Munro, Coleman and Cairns. This event, however, is not of very great significance, since the paresis disappeared after a short while, although Olivecrona and Ombrédanne saw permanent facial paralysis following the operation.

Much more important is the danger of a lesion of the cochlear nerve. At first, the division of the entire VIIIth nerve, *viz.*, of the vestibular and cochlear branches, was recommended as the proper treatment of Ménière's syndrome. In 1933, Dandy recommends that operation "because the patient loses nothing by section of the auditory nerve except the remains of hearing, which are of no practical value." It seems advisable ordinarily to save any hearing which is present, and it is a great disadvantage for the patient if he loses fairly good hearing by the operation. Such cases were reported by Crowe, McKenzie and Ombrédanne, and were probably not reported by others. On the contrary, Coleman and Lyerly say that almost all of their patients stated that they heard more distinctly notwithstanding the sacrifice of the auditory portion of the affected nerve, due most likely to the removal of the disturbing tinnitus in the affected ear. It is, of course, difficult to understand how one could notice improved hearing if the cochlear nerve is entirely severed.

Despite these miraculous results, we absolutely agree with Crowe, who claims that the ideal operation is one in which all vestibular fibres are divided and all cochlear fibres spared. Such an operation, however is difficult. Coleman and Lyerly believe it impossible. McKenzie, who studied the anatomy of the VIIIth nerve more thoroughly, found an absence of a fibrous septum dividing the vestibular and cochlear portion of the VIIIth nerve. He reaches the conclusion that it is not possible to accurately split the nerve; however, he believes that division can be sufficiently accurate for the present clinical purpose if the cephalad and dorsal half of the auditory nerve is severed. Cairns and Brain recommend division of the outer half of the auditory nerve, which is supposed to

contain the vestibular fibres. Dandy emphasizes that the hearing remains perfect or almost perfectly intact, if three-fourths, or even four-fifths, of the auditory nerve is divided. Ombrédanne cuts through the half or the anterior two-thirds of the VIIIth nerve in order to save the cochlear branch. Olivecrona recommends cutting through the vestibular branch in the middle third of the VIIIth nerve where the cochlear portion lies behind the vestibular branch, the latter partially covering.

It is obvious that there is no method of exact division of the vestibular nerve alone, and even in those cases in which the two nerves seem to be separated at operation an anatomical examination would probably reveal that this division does not actually hold true. Also, Dandy seems to agree with that statement, since he writes, under Fig. 2 of his paper in 1939, that the division of the anterior half of the nerve leaves the auditory branch "intact or practically so." We admit that microscopically the cochlear and vestibular branches often can be distinguished, since the myelin sheaths of the vestibular branch are, as a rule, thicker than those of the cochlear branch, as was pointed out by Alexander many years ago. Furthermore, the cochlear nerve contains the glia septum of Alexander and Obersteiner.

But as far as surgical anatomy is concerned, that division is not possible, since particularly the N. sacculoampullaris often lies almost within the cochlear nerve. Consequently, if a neurosurgeon claims to cut the entire vestibular nerve, it appears to me that he says more than he is actually able to do, since he seldom can know exactly how many fibres he has cut. This uncertainty explains the cases in which the labyrinth responded to the caloric test, despite the division of the "entire" vestibular nerve, the cases in which the hearing was saved despite the division of the "entire" VIIIth nerve, and finally the cases in which the function of the labyrinth and the cochlea was lacking after the division of the vestibular nerve alone. Many neurosurgeons, therefore, emphasize that for practical purposes, *viz.*, for the cure of the dizzy spells, the actual division of the entire vestibular branch is not necessary. In that case, the operation does not satisfy our pathologic criteria since an acute disease of one part of the semicircular canals or of one branch of the vestibular nerve is not known.

All these arguments are not supposed to deprecate the value of the operation. They only emphasize the strict indications for operation, a point on which a great number of neurosurgeons agree. We are, of course, far from pretending that the conservative treatment is an ideal one, although we do not agree with Cawthorne and Fawcett, who consider the dietary treatment as only palliative, but never as curative. Nevertheless, it is necessary to consider the results of the conservative treatment with a great deal of thought, as shown by the following case which I observed in Dr. Lederer's department at the University of Illinois:

A white woman, B. G. McG., age 59 years, had a Menière's attack in December, 1937, for the first time. Four months later she had a second attack. Subsequently, all teeth were removed. However, in September, 1938, she again had an attack, and these now followed at short intervals. In October and November, 1938, she had an attack every third day, which even awakened her from sleep. Every attack was ushered in by tinnitus and followed by pain in the right side of the stomach. She was never unconscious during an attack. On Dec. 12, 1938, we found retracted drums on both sides and atrophic scars in the anterior inferior quadrants. She heard accentuated whispered voice on the right, 6 feet; on the left, 10 feet; conversational voice, 18 feet on both sides. Weber was lateralized to the left. Rinné was positive-negative on the right, positive on the left; Schwabach was normal on both sides, c_4 was shortened on the right for 9 seconds, on the left for 6 seconds. She had a slight spontaneous nystagmus to both sides, and the caloric test was normal on both sides. On Dec. 13 she had a Ménière's attack, with a very marked nystagmus to the right, pastpointing and falling to the left. During the attack she heard accentuated whispered voice on the right side for 1 metre; on the left the hearing was normal. She felt better lying on her right side. On the evening of the same day, she had another attack. On Dec. 15, she again had an attack with severe tinnitus and dizziness, but on this occasion she had a nystagmus of third degree to the left and pastpointing to the right. Lying on her right side she felt worse, while on her left side she felt better. She heard accentuated whispered voice on the right side for 1 metre. The general examination revealed no signs of general arteriosclerosis, and her Wassermann was negative.

In searching for a cause of her Ménière's attacks, gallstones were discovered, and she was operated on for cholethiasis in December, 1938. After the operation she felt much better, and never had dizzy spells again. The tinnitus was markedly improved. However, she began to notice pain in the occiput, which grew gradually worse. She was re-examined on Aug. 11, 1939, and the otological examination revealed essentially the same findings as in December, 1938, except that the Weber was not lateralized, c_4 was shortened for 5 seconds on the right, for 3 seconds on the left side, and there was a spontaneous nystagmus of first degree to both sides. The fistula test and bilateral calorization did not give response.

During her stay in the hospital she passed through a period of severe excitement, and following that, on Aug. 14, had her first attack since her abdominal operation. This, however, was different from the previous spells. She had no marked dizziness nor tinnitus, but her pain in the occiput increased and she had a nystagmus of third degree to the right. Despite the nystagmus, she could rise from the bed without help, and she could even walk, with but a slight tendency to fall to the left. On Aug. 15, the attack had ceased but she still had a rough nystagmus of

first degree to the left. On Aug. 17, she only had a slight nystagmus of first degree to both sides. On Aug. 24, she was dismissed from the hospital without having had another attack.

In this case the removal of the focus in the gall bladder had a definite influence on the Ménière's syndrome. This lasted for about eight months. In the meantime her complaints changed. No more did she complain of dizziness and tinnitus. The pain in the occiput possibly indicates that we have to deal in this case not with an idiopathic but a symptomatic Ménière's syndrome, although it was not possible to discover the etiology.

A further disadvantage of the conservative treatment, especially dietary, is the necessity for hospitalization of the patient. Mygind and Dederding, as well as Furstenberg (quoted after Crowe), emphasize that fact and keep the patient in the hospital for eight to 10 days. Finally, Olivecrona finds that some patients are very much disturbed, especially by the dietary treatment.

Summarizing, we must say that at present the following procedure seems to be advisable: In every case of Ménière's syndrome it must first be decided whether one has to deal with a symptomatic or an idiopathic Ménière's syndrome. There is no doubt that with increasing accuracy of the general examination, the number of cases of idiopathic Ménière's syndrome will decrease. If after thorough examination the case has to be considered as idiopathic Ménière's syndrome or as a symptomatic Ménière's syndrome with an intractable pathology (*i.e.*, Paget's disease), conservative treatment must be installed.

As far as dietary treatment is concerned, I myself have no experience. It should be tried if the patient can afford hospitalization. The administration of bromides, iodine preparations (*i.e.*, sajodin) and calcium preparations (*i.e.*, atrocal or afenil) in more advanced cases can be recommended. Furthermore, I still use Faradization of the ears. I do not dare to decide whether or not the electric current works by suggestion. Certainly, in some cases the patient feels improved after the treatment without asking whether he owes that improvement to the electric current or to suggestion.

As far as catheterization of the Eustachian tube is concerned, I make an attempt in every case, even in those with normal drums. It is an old experience, emphasized particu-

larly by Bezold, that there are chronic adhesive processes of the middle ear which are associated with an entirely normal drum. Following our experience in the pathology of the ear, we suppose that in these cases the adhesions are situated within the niche of the windows only, thus not affecting the drum at all. The diagnosis in these case can be made: *a.* from the X-ray picture of the mastoid process, which shows, as a rule, an incomplete pneumatization; *b.* from the drum, which, although normal in general, often shows the signs described by Wittmaack as being significant for an incomplete pneumatization; and *c.* from the fact that the patient may have a definitely retracted and dull drum on his other side. Since Bezold has already emphasized the fact that chronic adhesive processes of the middle ear, combined with a normal drum, very often bother the patient more than those with a markedly retracted drum, the catheterization in the former cases is even more indicated. Unfortunately, there are some cases of Ménière's syndrome which simply cannot bear the mechanical treatment of the ears. In such cases, of course, that treatment must be omitted.

There is no doubt that obvious foci of infection in the body must be eradicated. In that respect I again wish to call attention to foci within the abdomen as exemplified in the above case, B. G. McG. If the conservative treatment has no effect at all, or if the patient is not willing to stay under observation for a long period of time, the division of the vestibular branch of the VIIIth nerve comes in for consideration. But even under these circumstances we do not recommend the operation in every case. In my opinion there are particularly two conditions in which the operation should be postponed as long as possible: *a.* if for another reason the patient is deaf on the side which *is* not affected and has some remains of hearing on the affected side, and *b.* if the hearing on the affected side is good.

a. If, *e.g.*, the patient has a cholesteatoma on the left side, which made him stone-deaf on that side, he later becomes ill with Ménière's attacks from a chronic adhesive process on the right side, the nerve should not be divided on the right side, even though the hearing on that right side is poor. That is obvious when one considers the possibility that in an operation on the right side the cochlear nerve may be damaged and, consequently, the patient becomes completely deaf on

both sides. *b*. If a patient has Ménière's attacks and good hearing on the affected side, the operation can be recommended only if the patient knows the risk concerning his hearing and is prepared to take it. The majority of neurosurgeons agree that not every case of Ménière's syndrome should be operated upon. Well proven indications and contraindications increase the value of the operation; enthusiasm decreases it.

I very well realize that this paper is filled with unsolved problems; however, the scientific interest in medicine depends on its unsolved problems. In this respect otology is very interesting, and one of the most interesting problems is Ménière's syndrome.

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307 North Michigan Avenue.

THE USE AND EFFECTIVENESS OF HEARING AIDS.*

DR. GORDON BERRY, Worcester, Mass.

I.—INTRODUCTION.

A major part of the time of the members of the American Otological Society is devoted to the cure or alleviation of impaired hearing. We strive with every skill and agency at our command to anticipate any tendency to deafness, rectify near or remote causative factors, eliminate aggravating pathology, and so treat and guide our patient as to cure or minimize the malady of deafness. When these measures have been carried out, what then? Formerly we had to tell our patients: we have done our best; we have no more to offer; good-bye. So started the dizzy round which took earlier unfortunates from doctor to doctor, then to the charlatan, and finally into isolated despondency.

In 1919, it became my privilege to see the remarkable accomplishments with lip-reading among the war-deafened soldiers at Cape May.[1] It was a natural step from this interest to an active participation in the national work carried forward by the American Society for the Hard-of-Hearing.[2] Here, a buoyant membership, largely composed of those pronounced hopeless by the otologist, were actively participating in a national philanthropic enterprise. And it was the two agencies of lip-reading and hearing aids that had made their participation possible, returning them to a life of happiness and usefulness. We have advanced since that day to a point where all leading otologists are conversant with those two constructive helps and recommend them to their hard-of-hearing patients. But are we convinced as to their efficacy? Are the hard-of-hearing who know the virtues of these aids remiss or laggard in presenting them? Are the manufacturers at fault? Do otologists make themselves sufficiently conversant with them? It is these and similar queries that prompt me to present this subject of the use and effectiveness of hearing aids for your thoughtful consideration. It may hap-

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pily include three viewpoints: that of the physicist, that of the user, and that of the otologist. The writer's deafness has familiarized him with the user's viewpoint, his otologic practice, though representing a limited experience, gives him a background for presenting the otologist's viewpoint. He is not intimately conversant with the engineering principles embodied in hearing aids. He must look to the physicist to tell him their effectiveness, and how that effectiveness may be best applied. To this end, he has written to medical and to physicist friends, also to the manufacturers of many of the hearing aids. Their generous response has made possible the assembling of much of the material in this paper. In an effort to acknowledge their courtesy, I am listing them with the references at the end.

II.–THE PHYSICIST.

A—Sound Considerations.

1. Sound Intensities and Frequencies Increase in Logarithmic Ratios:

Let us first turn our attention to the physical aspects of hearing aids. An early difficulty comes in comprehending the logarithmic values employed. For instance, telephone research workers discover that a sound painful to the average normal ear in the middle register is one million million times as loud as a sound that is barely audible. This is multiplying by 10 to the twelfth power. They call this 12 bels, each bel increase representing a magnification of the given sound 10 times. For finer differentiation, each bel is divided into 10 dcb. An easier way to comprehend decibel values is to consider the sound intensities employed in conversation. Average speech power uses about 60 dcb. of noise. Loud speech uses 80 dcb. This is 20 dcb. louder, or 100 times as loud. Weak speech would use 40 dcb., and a soft whisper 20 dcb. The difference between a soft whisper and loud speech is 60 dcb. How much does this mean? Sixty decibels or 6 bels means 10 to the sixth power, or the addition of six ciphers. Then, loud speech is 1,000,000 times as loud as a soft whisper.

Musical frequencies also change in logarithmic ratio. The usual audiometer range is seven octaves, each higher octave showing twice as many frequencies as its next lower. So we

multiply the bottom pitch at 64 cycles by two to the seventh power and secure the top pitch figure of 8,192.

2. Intensities of Individual Speech Sounds:

We have noted the great variations between loud and soft speech intensities. There is a similar divergence in ordinary, quiet conversation. Accented syllables are three or four times louder than the others, vowel sounds are louder than consonants, and the different consonants vary greatly. The Ewings,[3] of Manchester, England, find the intensities used in quiet conversation ranging as much as 56 dcb., making the loudest sound 500,000 times as loud as the weakest. Their analysis of these fundamental speech sounds is pertinent. Considering the greater intensities first, the ó in "aw" or ah in "balm" are the loudest speech sounds. In quiet conversation, their loudness level above the normal threshold of audibility is 60 dcb. All but one of the vowels and two of the consonants, r and l, are included in the intensity range, 60-50 dcb. One vowel sound, ēē, and all but the weakest consonants appear between 50 and 40 dcb. And the weakest consonants, p, d, b and th range from 40 to 38 dcb.

3. Prevailing Noise Values:

Harvey Fletcher's monumental treatise on "Speech and Hearing,"[4] illustrates in another way the relative amplitudes of the usual sounds by telling us the masking effect of these noises in the following chart:

Amount of Noise in Dcb.	Typical Place for Such Noise	Maximum Distances for Hearing Average Speech in Such Noise
0 Dcb.	Soundproof booth	1250 feet
10 Dcb.	Country residence	395 feet
20 Dcb.	Quiet office	125 feet
30 Dcb.	Average office	40 feet
40 Dcb.	Department store	12 feet
50 Dcb.	Train or automobile	4 feet
60 Dcb.	New York subway	15 inches
70 Dcb.		5 inches
80 Dcb.	Boiler factory	1.5 inches

4. The Effect of Noise and Distance on Hearing Aid Efficiency. a. Sound Intensities:

Note in passing why Mr. Jones with a 40 dcb. loss in hearing has no handicap in a railroad car; for the prevailing

noise of the train masks Mrs. Jones' normal ear 50 dcb., and conversation to be heard by either must be raised well above this level. Now let us follow them to their home, where the masking noise may be 30 dcb. They are talking in the library. The 10-foot space between them attenuates their voices 40 dcb. more, making a total muffling handicap of 70 dcb. Under such conditions many speech sounds are scarcely heard by normal Mrs. Jones. What about her husband, with his 40 dcb. deafness? The 30 dcb. noise of the room does not bother him. But the 10-foot distance with its 40 dcb. handicap must be added. This places his threshold of hearing at 80 dcb. Any sound to be clearly distinguished must be at least 10 dcb. above this threshold, or 90 dcb. It takes no imagination to realize how broken and inarticulate his wife's conversation must seem.

The acoustic engineer knows with precision the sound intensities of speech. The audiogram gives him the decibel loss in hearing. He knows the masking effect of differing environments. Newhart and Hartig[5] showed graphically at the last Academy meeting how he must now fashion a hearing aid with definite amplitudes to overcome these handicaps, thus bringing the known sound intensity through the known masking noises and over the known hearing loss barrier, direct to the acoustic nerve of the listener. This lends itself to mathematical exactitude.

Can one use these exact figures and, putting them together, determine just what hearing aid the patient requires? Unfortunately, the answer must be in the negative. The trouble is not with amplification *per se*. If we disregard such items as size, expense and electricity consumed, the sound can be amplified almost indefinitely. The chief hindrance comes in our inability to bear these amplified noises. We sometimes forget that the upper sensation limit where pain begins is the same whether the ear is normal or deafened. So the band or belt of hearing available for our patient, instead of the normal 130 dcb. width at 1,000 cycles, may now be narrowed to 20 dcb. Experience has taught the Ewings they cannot amplify above 110 dcb. or the louder accented sounds of speech become too painful. Now we begin to appreciate how difficult it is to make a hearing aid strong enough but not too strong.

b. Sound Frequencies:

We have been considering noise intensities. In a somewhat similar way, pitch frequency variations and responses may be analyzed. Here we run into the many variables of speech. In approximate terms, singly resonant vowels (such as ū and ō) range below 1,000 cycles; while consonant sounds (such as k, t, f and s) range above 2,000 cycles. The normal hearing range approximates 20-20,000 cycles.[6] Normal speech may be said to range from 90 to 8,000 cycles. These are the basic frequencies we are dealing with. Other illustrative ranges are: the piano reaches from about 28 to 4,200 vibrations per second; while a man can whistle from 400 to 4,000.[7] All mechanical contrivances for amplifying sounds exert a narrowing effect, in that they permit only a limited range of these sound cycles to pass through. For instance, the sound vibrations coming through the ordinary telephone system are limited to 300-2,500 vibrations.[8] This is adequate for the normal ear, for the most important speech frequencies are between 500 and 2,000 cycles.[9] The telephone relies on carbon granules for sound conversion. The radio and auditorium sound amplification devices offer a wider range of 200-3,500 cycles. These use vacuum tubes for sound conversion.

B—HEARING AID CONSIDERATIONS.

1. Carbon Granule vs. Vacuum Tube Microphones:

Hearing aids relying on electrical power for amplification are of these two same types: the carbon granule type and the vacuum tube type. The English[10] call these the nonvalve and the valve type, respectively. I will call them the carbon aid and the vacuum aid. Both have been in use for some time. Only recently, since very small vacuum tubes have been made available, has it been possible to make the vacuum aid small enough to be wearable. At first its application was limited to stationary or desk sets. Later it could be carried in a box and was called portable. Briefly, the relative advantages and disadvantages of these two types may be listed as follows:

a. Types Contrasted:

1. Disadvantages of the Carbon Granule Type of Hearing Aid:

a. Limited magnification frequency (300-3,000 cycles).

b. Nonlinear distortion (*i.e.*, amount of sound reaching the ear not proportional to that striking the microphone).

c. Uneven amplification, making peak sounds too loud when other frequencies are loud enough.

d. Over-emphasis of background noises.

e. Less applicable for nerve deafness forms because of these four faults.

f. Tendency for the granules to become fixed, requiring a jarring loose for them to function at maximum efficiency.

g. Microphone must be upright to function, though this is being corrected.

2. Advantages of Carbon Aid:

a. The instrument is smaller and lighter.

b. It has had time to be better perfected, and by the larger, stronger companies.

c. Less electricity consumption. Cheaper to use and to repair.

d. It has proved adequate for lesser degrees of deafness (20 to 60 dcb. loss).

3. Disadvantages of Vacuum Tube Type of Hearing Aid:

a. It is a heavier, larger instrument, though much smaller during the past year, and others are soon to come on the market.

b. The present instrument is of recent manufacture and has not had time to be developed as well.

c. It is more expensive to buy, and apparently more expensive to run because of greater electricity consumption and more costly repairs.

4. Advantages of the Vacuum Aid:

a. Permits magnification in all speech frequencies.

b. Is free from distortion and permits even amplification. Through the use of filters, over-amplified frequencies which are too loud may be cut down.

c. The resulting selective amplification makes this type peculiarly fitting for severe deafness (more than 50 dcb. loss), and especially for nerve deafness (marked loss above 2,000 cycles).

d. Equally efficient at all times and in all positions.

5. These faults may be summarized by saying that the carbon aid is more standardized, is smaller and lighter and cheaper; while the vacuum aid is more powerful, permits selective amplification for varying forms of deafness, and as its manufacture becomes increasingly perfected and refined it may supplant the carbon aid. Other recent forms of commercial acoustic amplification have shown a preference for the vacuum tube.

b. Types Discussed:

Physicists differ as to the respective values of these two types. The writer from the Sonotone Corp.[11] is in a position to express an impartial view, for his company manufactures both types. He explains that the tube offers a wider range, or frequency band, but that if the user is totally deaf to high frequencies, this extra amplification in the high tones is of no value to him. Another interesting difference is that the carbon aid deteriorates, gradually giving the user warning that the carbon elements need renewal; while the tube aid when the tubes give out (lasting four months to over a year) does so without warning and may leave the user high and dry at a crucial time. Both types permit the desired amplification. He considers the chief advantages of the tube aid to be the great reduction in distortion, while its chief handicap is the greater cost of operation. This cost appears in replacement of worn elements and in battery consumption.

The Radio-Ear Co.[12] seems to agree with this. It, too, has developed both types. Their own experience, and their review of the instruments made by others, led to the conclusion that they can offer more hearing per dollar of maintenance in the carbon aid. Continuing research has eliminated some of the undesirable characteristics and has led to their present emphasis on the carbon instrument. The advantages of the vacuum aid are appreciated; it is the cost factor that discourages its general use. The Aurex Corp.,[13] which is now

putting out a new wearable vacuum aid, questions this general criticism of the added cost. Through special effort they have been able to reduce their battery consumption to at least one-half, putting it on a par with that of a carbon aid.

One trouble in the carbon aid has been that the customary earpiece has dampened out some of the higher frequencies reaching it. The Western Electric instrument[14] has improved this phase by an electrical stepup in the earpiece of their "orthotechnic" model.

Though Sonotone claims to have put on the market the first portable vacuum aids, and though other eastern companies (Dictograph Products, General Sound Product and Radio-Ear) are now manufacturing them, the western groups (Aurex, Maico, Telex, Vaculite, Wengel) seem to be more active in advocating them. Knudsen[15] joins in this, feeling that their future possibilities and development are more promising, and that they lend themselves to greater amplification and more exact fitting than the carbon aids do. Claims as to the amount of amplification possible in the wearable vacuum aid vary from 40-70 dcb.

All these companies furnish sales literature, most of it of little help to us. The Maico Co. gives a chart showing the high efficiency of the carbon aid at its middle peak with sharp drops on each side, while the vacuum graph gives a more even gain, spread through all the frequencies. An excellent pamphlet entitled, Hearing Aids,[7] has just come from the Sonotone Corp. The Wengel Laboratories sent me a helpful pamphlet entitled, Auralometry Explained.[8] These are worth while contributions and will be appreciated by otologists.

2. Amplifiers:

a. The carbon aid. The carbon granule chamber acts as an amplifier in the electric circuit. The sound-receiving carbon diaphragm of the microphone transmitter rests against carbon balls held in cups or wells in an abutting carbon block. When sound waves strike the diaphragm, they press it against the enclosed carbon balls in varying degrees. When the loose carbon balls are thus pressed firmly together, they become more permeable to electricity. These parts are so connected with the electric battery that there is a constant flow through

these carbon components. The increasing and decreasing pressure on the carbon granules hastens, then inhibits the current flow, giving us two factors: an oscillating current and a trigger control. The oscillating electric waves are transmitted back into sound waves in the earpiece or receiver. The trigger action, plus the electric current to furnish power, is the agency for stepping up the sound.

A so-called amplifier or booster is inserted into the existing circuit when greater sound magnification is desired. This is a second carbon granule system, similar to that in the microphone transmitter. A second trigger effect is thus secured. The end-result is more sound intensity at the expense of a little more current from the battery.

b. The vacuum aid. The amplifying agency in the vacuum aid is the vacuum tube. It is well illustrated and described in the pamphlet on Hearing Aids.[16] These tubes furnish the amplification for our radio sets and for the large type hearing aids. Only recently, and first in England, have the tiny vacuum tubes been available for small hearing aids. Each vacuum tube exerts a similar trigger action to that in the carbon amplifier but is free from the distortion and resonance factors obtaining in the carbon sets. As the sound potentials progress through the electric system of the hearing aid, they may be triggered up by the simple addition of more vacuum tubes and by the consumption of a corresponding increase in electric current. The resulting increase in power permits the use of the less efficient but better quality crystal transmitter in the place of the more efficient but less true carbon unit.

One company[17] now offers a continuously variable frequency selective amplifier to permit individual adjustments throughout the ranges of 50 to 7,500 cycles. The stationary set is in production; the wearable set is soon to appear.

3. Ear Terminals. Bone Conduction vs. Air Conduction:

Another major point of difference in hearing aids is the bone conduction versus the air conduction earpiece. The Sonotone Co. was the first to produce the bone conduction terminal, and stressed it heavily among their sales agents. Their recent circular says they have perfected a new air conduction piece. They offer the following general rule:[18] In terms of the audi-

ometric curves, if the bone conduction loss is less than half of the air conduction loss, a bone conduction hearing aid should probably be used, while if the bone conduction loss is more than half the air conduction loss, an air conduction hearing aid will probably be the more satisfactory.

Watson[19] discovered the frequency range for bone conduction to be almost as wide as that for air conduction, and ample to include the frequencies and intensities of speech sounds.

Jones and Knudsen[20] give helpful rules for prescribing hearing aids. If the hearing loss is 25-50 dcb., they prefer the carbon aid. If it is over 50 dcb., they advise a vacuum aid. If the impairment is primarily conductive, a bone conduction type of instrument is indicated; if it is primarily perceptive, an air conduction type.

Most of the manufacturers have emphasized the air conduction earpiece. There is considerable difference in performance, and it is natural that each group should emphasize that terminal giving them the best performance. My personal feeling is that bone conduction has been over-emphasized.

a. Sound pressure factors. One writer[21] feels that the bone conduction terminal serves in a higher percentage of carbon aid users because the terminal and the bone absorb the shock of loud sounds at "peak" frequencies which the eardrum cannot bear. But low intensity sounds have more difficulty in pushing their way through by the bone conduction route. The result is that vowels and low-pitched or loud intensity sounds pass through well by bone, while the finer sounds of higher frequency pass through better by air conduction. It is help in the latter that most users desire. He finds that air conduction lends itself even more to vacuum tube aids which amplify better than the high frequency sounds.

b. Distortion. If the deafness is not too profound, sounds come truer by air conduction. In an auditorium where both bone and air conduction terminals are available, I have found that the music of a singer or of the orchestra sound truer and less distorted by air conduction.

c. Gain from uncovering better ear. On the advantage side for the bone conduction terminal: if one ear is very deaf, the other and better ear would be the one used for an air conduc-

tion earpiece. This is all right when the current is on. But when the current is off (to conserve the battery), this better ear is plugged and the earpiece must be removed to permit this better ear to function; while a bone conduction terminal can be worn behind the ear (or on the other side when both bone conductions are good), and the better ear is at all times ready to function to the best of its limited ability, whether the current is turned on or off.

Here may be interpolated Hayden's advice[22] on which ear to use in the case of an air conduction terminal.

1. If the difference in air conduction is greater than 20 dcb. and the difference in bone conduction is less than 10 dcb., fit the ear with the greater loss for air conduction.

2. If the difference in air conduction is less than 20 dcb. and the difference in bone conduction is greater than 10 dcb., fit the ear with the smaller loss for air conduction.

This offers a guide. In practice, the user desires the best results obtainable. In my own case that has meant place the terminal in the better ear.

d. Appearance factor. An important point is the outward appearance. A lady can hide the band and mastoid terminal under her hair and put microphone and battery under her dress. A man's hair (or absence of hair) does not permit such concealment and, from the standpoint of looks, he prefers the midget air conduction earpiece. There is a third type of instrument, carried in a small bag or box. Here the terminal is preferably the air conduction one, for it is more easily slipped into and out of the ear than the cumbersome and awkward headband. Ladies with advanced hypacusia prefer these.

e. Fitted air conduction ear terminals. The shape of the bone conduction terminal is suited for almost any person, the adjustment necessary being chiefly in the tightness and spring of the headband. But the air conduction terminal deserves careful fitting. They are of two types. One is in the form of a flat diaphragm held against the outside of the ear with a retaining headband. This is the less efficient. The source of sound is farther from the ear drum and there is too much leakage of sound. The other is a small or midget receiver

that is inserted into the concha and external meatus. It may be of soft rubber, or of some hard material, as vulcanite, or a synthetic crystalline preparation. The soft rubber is not as cleanly but it stays in better unless it is a good fit, in which case a hard and smooth earpiece is much to be preferred. The best is this hard terminal, carefully made for the individual user's ear. The snug fit serves the threefold purpose of better hearing through no loss by leakage, of greater comfort in a tender ear, and of staying in firmly. I would recommend that the patient secure an individually fitted terminal when once he has made the final selection of the instrument.

This fitted ear terminal is made by the manufacturer or some agency serving for him. It requires a plaster model of the user's ear. Some manufacturers describe how to make such a plaster model, or directions may be learned from an article in the *Volta Review*.[23] A local dentist may be called on to make it. I feel that the otologist is the one best qualified. The procedure requires care as to detail but is otherwise simple. The hairs in the selected ear canal are trimmed, the skin is cleaned and greased, the bony canal is packed off with cotton, and a careful plaster model is made of the concha and cartilaginous canal. The earpiece made from this plaster model should fit snugly. Pressure points may need to be filed down.

4. Batteries:

a. Types of batteries. Battery consumption represents a continuing and considerable expense. In wet cell batteries, expended current can be rebuilt by recharging. These are being perfected and are preferred where a maintained high voltage is desired.[24] Some stationary sets are made to derive their energy direct by plugging in on the city current. But for portable and wearable sets, dry cell batteries are generally used, and these wear out. Manufacturers are trying to increase their power and longevity, and decrease the size and expense. Some hearing aid firms urge the purchase of their own batteries as being better adapted for their aids, and they even make the terminals unique so that other batteries will not fit. One suspects that the income derived from this practice is one argument for its continuance. These special batteries offer about 10 per cent more life.[25] Their smaller size and weight add to their attractiveness. Commercial batteries

recommend themselves to the user because they are so easy to secure. Now that the excellent Burgess batteries are being made so generally available, may we hope that the different companies will make terminals to fit either these or those of some equally good and well serviced concern?

b. Current consumption. Bone conduction terminals consume more current than air conduction, and vacuum aids consume more than carbon aids, according to most authorities. But some vacuum aid firms claim that research has brought their current consumption down to the carbon aid level. The vacuum aids use two types of batteries: the "A" battery is the simple flashlight cell used in the carbon aids, carrying a low voltage of 1.5 to 4.5, for furnishing current to the tube filaments. The "B" battery supplies 30 to 45 volts for the plate current. One firm[26] has reduced their battery operating cost between one-half and two cents per hour of use. Another firm[27] expects their "A" battery to last 15 to 24 hours, at a replaceable cost of 10 cents each; and their "B" battery to operate upwards to 300 hours, with a replacement cost of $1.90. Some users employ special current rechargers on their "A" batteries with questionable success, and others keep an extra supply and rotate their use, for the periodic rest prolongs their total life. The "B" battery does not lend itself to such efforts to prolong its life.

5. Initial Expense of Hearing Aids:

The approximate expense of these better aids ranges from $75.00 to $200.00. The vacuum aid averages higher. Cheaper aids are available but they do not seem to furnish either the satisfaction or the service. One is advertised for $30.00,[28] which I am hoping to learn more about. When the great and necessary overhead expense of research and sales propaganda has been met and when their use is more general, these instruments should be much less expensive. In the meantime, more than one company[26, 27] is offering to deal direct with the otologist, and another[21] offers a considerable reduction if the middle man expense is thus saved.

6. Personal Experience with Carbon and Vacuum Aids:

Learning of my interest in vacuum aids, the Aurex, Maico and Telex Cos. have kindly sent me trial instruments adjusted to my charted hearing loss. This has permitted me to compare

the two types, for my aid is of the carbon type, being an experimental model that Harvey Fletcher generously wanted me to try, but never would accept its return. It has served me so well that I still wear it, even though my loss for high frequencies is now much greater.

In making such a comparison, one must remember that there is a process of adjustment[29] resulting from the use of a hearing aid. Even though the sounds through my own aid are distorted, they have come to seem normal to me. Whether or not one has used a hearing aid, the sounds coming through a newly tried instrument will probably seem strange and distorted, because they differ from his accepted normal. So the user may not be the best judge in the selection of a new aid. A conscientious trial is necessary to permit a true evaluation.

In my case the loudness of the more accented sounds seems but little greater with the modern vacuum aid, over my own now obsolete instrument. But the fine consonant sounds come through much more clearly. This permits greater comfort in listening when in a quiet environment, through a rheostat lowering of the sound intensities throughout. For, in my carbon aid I have to stepup the rheostat almost to the painful point for louder sounds, in the effort to hear the faint consonant sounds; though usage seems to make it easier to bear these louder intensities. The added handicap of a noisy environment will be elaborated later.

7. Standardization of Hearing Aids:

The Committee on Deafness Prevention and Amelioration, with Dr. Horace Newhart as Chairman and an appointed membership from the national ear societies, has urged impartial and expert reviewing of commercial hearing aids in order to protect the purchaser. This is now being done effectively by the Council on Physical Therapy of the American Medical Association, through its Committee on Audiometers and Hearing Aids,[30] Dr. Austin Hayden, Chairman. The immediate difficulty is that the different research laboratories are turning out new models so rapidly. It takes time to review carefully and accept or reject an offered model.

Further help is available in a recent interesting series of articles[31] on Hearing Aids in the *Volta Review* by the editor, Josephine B. Timberlake.

C—SUMMARY.

The otologist does not aspire to be an acoustic engineer. But a realization of the wide range of sound intensities and frequencies in speech seems essential if he is to appreciate clearly how his patient hears or why he fails to hear. The masking effect of surrounding noises troubles less as deafness progresses. Before the hearing loss becomes a handicap, the careful selection of a hearing aid should be recommended by the otologist who should familiarize himself with their salient characteristics.

The two major types are the carbon granule and the vacuum tube aids. Each presents advantages and disadvantages. Each has its own type of amplifier to stepup the sound. The vacuum aid can use crystal sets. Each offers a choice of a bone or an air conduction ear terminal.

The power is electrical, furnished by cell batteries or the city current, in the stationary sets; and by wet or dry cell batteries in the portable and wearable sets. In the vacuum aids, the battery consumption has represented a considerable expense, which research engineers are trying to cut down. The American Medical Association Council on Physical Therapy is doing a valuable service by examining these instruments and reporting on their merit.

III.–THE USER'S VIEWPOINT.

The user's viewpoint would properly include a consideration of the many psychological aspects[32] which time and space will only permit us to touch upon. Here we may consider three phases or periods: the first, that prior to his using a hearing aid; second, while he is using one; and third, when it is of little or no service to him.

1. Prior to Using a Hearing Aid: Almost without exception, the hard-of-hearing patient needs a hearing aid before he seeks one, or will accept it. Yielding to the pressure of his desire to postpone, the otologist, too, delays unduly before urging it. Such delay is natural. The patient wishes to hide his infirmity. This is a human trait, and an honorable one. At first, deafness lends itself to such deception, for there is no external evidence of the malady. And even the cripple is

commended when he trains himself to show as little of his limp as is possible. So the patient holds onto the idea that he still hears, much as a man at threescore years maintains that his youth and vigor are not diminished. Neither is honest with himself or with his neighbor. The otologist will save his patient much sorrow, and the family and friends much hardship if he persuades every patient with a demonstrable hearing handicap, whether in his social or in his economic life, to use a hearing aid. The point at which this handicap begins is a variable one. Hearing aid serviceability begins at around 25 dcb. loss and ends at around 85 dcb. loss.[23] If the patient cannot hear the lecturer, and can do so by moving up front, the handicap need not yet exist in that particular environment. Actually I have known a patient to stay back and hear but little, because the very act of taking a seat farther forward would be to confess to himself and to his friends that he was slipping. To him who understands, here is the first chapter of a tragedy which need not be written if a wise otologist intervenes. Last month a personal friend (not a patient) confided that once she had actually tried a hearing aid in a church pew where she chanced to sit. It had been a real effort for her. She knew she was hard of hearing but was sure almost no one else knew it. Never, never would she permit herself to wear one. As she talked to me, I sensed that there were two fears pressing on her in that church pew: one that someone would see her trying the hearing aid, and the other the fear that it really would help, thus putting her in the wrong the longer she refrained from accepting one.

The actual decision to accept a hearing aid is a more significant act than most otologists realize. It seems easier to get the patient to try one if one can avoid making a major issue of it. Pressure urges him to build up his defenses. Make it informal, minimize the importance of this first step taken. A trial before purchase helps. Tell him to get one for only occasional use: at lectures or in the theatre, where a little more hearing will be such a help; not to bother with it at other times. If the patient is a woman, perhaps she would like a cute little box or a bag on her arm, from which she can slip a lorgnette-like earpiece to use when the speaker mumbles his words. For, remember: at this stage, it is the lecturer's poor diction that is to blame, or the bad acoustics of the hall, certainly not your patient's deafness. A visit to a league

for the hard-of-hearing accomplishes much. Here she sees the members using hearing aids without embarrassment and without concern. It is the natural thing to do. She enjoys a lecture through the group hearing aids there available. She discovers that each wearer takes delight in having an earphone, is grateful for the freer life and the broader horizon that it permits. So, lead rather than drive the patient over this first hurdle. Make it seem low and easy to jump.

2. While the Patient Is Using One: This period embraces the one when the patient frankly accepts his hearing handicap and secures a hearing aid. What is the first reaction? I think many parallel mine. I was frankly disappointed. I had postponed its acceptance, but with an inner assurance that if and when I did consent to this stigma I would enjoy the reward of nearly normal hearing. This was far from true, and is the more true the longer the acceptance is delayed.

When once I had accepted this disappointment, my next trouble was in wearing it. I felt conspicuous. Particularly, children wanted to know what that box thing was for. It was a continuing effort, gradually conquered, though never completely, for me to wear it. I think the strongest argument persuading me to use my earphone at home, or in committee meeting, or in group conversation, was the realization that I owed it to my friends to help them all I could if they were willing and desired to talk to me. What right or courtesy could there be in making them talk loud for my sake, when the hearing aid could spare them this effort!

Most of such cases become deafer. We of this generation are favored here. The designing and manufacture have steadily improved. Many who cannot now gain sufficient amplification with the models that once served them do secure the required help with these modern, better instruments.

There is an occupational phase which is not emphasized enough. A progressive deafness may make the patient's employment precarious. The usual attitude of a hard-of-hearing salesman is that the wearing of an earphone would advertise his handicap and create a barrier between him and his prospect. I think experience proves the contrary. The prospect asks a question and gets an erroneous reply. He concludes that the salesman is stupid or that he is deaf, and their relations become strained on either count. When the sales-

man wears his earphone and, furthermore, gives the right answer, the prospect knows the salesman is not stupid, is honest with his environment, and that the earphone makes him efficient. The first contact is auspicious. Those who are interested in this occupational phase may wish to read the results of the occupational survey made by the Federal Government[34] or my report of one unit part of the Survey.[35]

3. When It Is of Little or No Service to the Patient: Lastly, under this heading of usage comes the time when the deafness may become so advanced that the electrical hearing aid is of little value. Or we may be dealing with a sensitive, retiring lady, who cannot become adjusted to being seen with one on; or the amplified and distorted sounds may be too unpleasant, even painful. If through practice or observation or training they have already learned lip-reading, they are, indeed, fortunate. Here is a strong argument for learning this art early, before it is imperatively needed. Or an ordinary speaking tube may be just the thing. With this, speech reaches the ear in its true values, not distorted. The listener hears the speaker's voice with the same intensity as if it were spoken directly into his ear. The mouthpiece should be held below the speaker's mouth to permit lip-reading at the same time.

4. It will be helpful to summarize the applicability of the average adjusted hearing aid to the user in terms of the amount of deafness present. I am indebted to Joseph B. Kelley[36] for helping me in formulating it.

20- 40 Dcb. loss gets much help from an ear phone, but prefers to manage without. Probably does not feel the need of lip-reading.

40- 60 Dcb. loss needs one, but tries to wait till the deafness is worse. This is the group that gains most from an electrical ear phone. Does not wear it constantly, but for lectures, church, theatre, group conversation. Air conduction is preferred to bone conduction. Lip-reading does not seem so necessary; would help greatly.

60- 80 Dcb. loss needs an ear phone for all conversation, but does not secure as much help as the preceding group. Bone conduction is preferred over air conduction. Lip-reading becomes more necessary.

80-100 Dcb. loss.—Gains less. Bone conduction is much preferred. Is so ineffective that lip-reading becomes imperative.

IV.—THE OTOLOGIST'S VIEWPOINT.

It has been pointed out that on the otologist devolves the double task of controlling the pathology insofar as his skill

will permit, and of urging such helps as lip-reading and hearing aids will afford. The lip-reading is handled by a trained teacher. Who will advise concerning the hearing aid? I submit that the otologist is in a strategic position to render this service.

1. Can a Hearing Aid Do Harm? At this point we may refer to the tradition that a hearing aid may injure the hearing. I think this idea results from the fact that once a user accepts a hearing aid, he discovers how much he has been missing and is no longer satisfied with his unaided ear. To himself he seems deafer than he used to be. In point of fact, the use of a hearing aid tends to increase rather than decrease acoustic intelligence. Disuse of any function encourages its atrophy. Using our ears probably does not improve our threshold performance, but it does make our acoustic perception keener. This has been demonstrated time and again. We do our patient wrong if we urge against his employing this means for keeping active his acoustic function. The only word of warning I would utter would be against too powerful an instrument in an advanced perception deafness case. The range between his threshold at the bottom and his sensation point at the top is very narrow, and it is too easy for the incoming amplified noise to reach painful proportions. When so amplified they parallel the noise of riveting or boiler-making, with the important difference that the user can cut off the noise at will. I have seen no concrete case showing nerve injury. But I am careful to warn my patient to turn down the rheostat control when the noise becomes excessive. I note that Fowler[37] agrees with me here. The Ewings[38] have never seen any ill effects in their large experience. They feel that rapid changes in noise pitch and intensity, coupled with the protection that the user's avoidance of pain automatically affords, prevent any danger from noise deafness.

2. The Otologist Guides in the Selection: It has been pointed out that a hearing loss of 20 to 40 dcb. is not profound, but that a hearing aid will help; that a light carbon-granule type instrument is preferable at this early stage; that if the deafness is progressive we should urge the patient to use an aid as occasion urges and so get accustomed to it, against the possible day when its use must be more constant. When the decision has been made to secure a hearing aid, it

has been my practice to give the patient a copy of his audiogram, with letters of introduction to the local agents, preferably more than one. He is to try their best selections, then take home on trial that instrument seeming to help best. It is important to try it out in the patient's normal environment, not in the artificially ideal surroundings of the agent's office. Before the final purchase he brings it to my office for my review.

3. Audiometer Test of Hearing Aid Efficiency: There are several ways for the otologist to discover whether the hearing aid selected is serving well. Hayden[39] has recommended a direct hookup between the earpiece of the audiometer and the microphone of the hearing aid. He is able thus not only to discover the hearing aid's performance, but he can tell wherein it is less effective, and what change in possible filtering and amplifying combinations should be made to secure an ideal result. Braly[40] supports him. I regret that I have not yet been able to secure any such happy results. Instead, I can hear my 2A Audiometer tone better with my unaided ear at most frequencies. The difficulty may be that the audiometer tests for sound thresholds, while my hearing aid operates best at higher amplifications, and in the middle pitch ranges. If I read them aright, Fowler[41] and Steinberg[42] support me here.

4. Graduated Vocal Tests Under Calibrated Control: Some recent makes of audiometers have a microphone attachment with calibrated control through which the tester can speak. The patient wearing his hearing aid, without the help of lip-reading, listens to the varying voice intensities and shows by his responses the efficiency of the hearing aid being tested. This is a good method but is abnormal, in that the customary noise environment is lacking.

5. Tests Under a Normal Noise Environment: Hearing aids should be tested in a varying background of noise. This is the normal environment in which they will be used. Kelley[43] points out that any accurate method for testing the efficiency of hearing aids would wisely provide such a noise background. Ideal test conditions would include a large soundproof, acoustically-treated room, into which could be introduced the controlled test tone through a loud speaker. At the

other end would be placed a calibrated hearing aid for control, and the instrument to be tested. Into this system can now be introduced known pressure factors, and with differing tone frequencies. The result would give exact information concerning each hearing aid tested.

A committee sponsored by the Acoustical Society of America and the American Standards Association has been trying to standardize methods of determining the response characteristics of hearing aids. There are as yet no common standards of evaluation. Each maker knows accurately the performance characteristics of his own hearing aids, but in terms that are not comparable with the terms used by other makers. Among other objectives the Committee is seeking a technique for measuring:

a. Response frequency characteristic.

b. 1. Variation of response with time. *2.* Variation of response with temperature. *3.* Variation of response with position. *4.* Variation of response with humidity.

c. Nonlinear distortion.

d. Background noise.

e. Current consumption.

f. Attenuation range.

g. Regeneration or degeneration.

Such a list gives us an idea of how complicated the problem is. Until more data are available, what tests can the otologist apply?

6. Simple Voice Tests for Office Use:

a. A quick voice test. If the otologist has no calibrated voice-producing mechanism, he can use what I have found helpful: a simple number test. Use first the essential vowel sounds (such as one, four, five), then the essential consonant sounds (such as six, seven). This is done in the office with either the whispered or with the spoken voice, depending on the degree of deafness being tested. The other ear and lip-reading are excluded. The number test is applied to the unaided ear, and then with the hearing aid at optimum full

strength, and the distances noted. The important part is to use the same voice intensity without and with the hearing aid, and in the same noise and room environment. This is grantedly a rough test, but it can be easily and quickly done, and does give an index as to the hearing aid response. I expect the aided ear to hear at least five times as far as the unaided ear. Some do much better. It should be remembered that these are artificially ideal conditions, and that the same hearing aid will not give such favorable responses in a normal conversation environment.

b. Nonsense word tests. It is recognized that the number test is inaccurate. There is too much chance for a quick imagination and a clever guesser. Sentence tests are the least inaccurate. Bell Telephone Laboratory tests[44] showed that only 30 per cent vowels and consonants need to be heard to give a 90 per cent score on sentences. For this reason, if the proportion of accurately heard sounds over a telephone system is 85 per cent, conditions are considered good, and 70 per cent is permitted as the minimum. Similarly, numbers offer a known pattern and lend themselves to guessing. Control tests have shown that a hard-of-hearing person may give a 90 per cent response to numbers when nonsense syllable vowels give 80 per cent and consonants give but 25 per cent.

In order to eliminate entirely the element of guessing, the Bell Telephone Laboratories used carefully selected one-syllable vowel and consonant words.[45] Ewing[46] uses what he calls a short intelligibility test on the unaided ear. He uses both numbers and nonsense syllables. He grades the cases into four groups, in accordance with the number of consonant sounds they answer correctly. He finds from experience that each graded grouping gives him a rough index of the type and degree of deafness, of the gain that may accrue from a hearing aid, and of the type of instrument to consider.

The *Volta Review*[47] gives a nonsense syllable test by Robert West, Ph.D., Professor of Speech Pathology at the University of Wisconsin. There are 25 pairs of syllables, arranged in five groups, each designed to emphasize different speech frequencies. The tester uses a natural conversational voice at four feet. He reads one syllable of each pair, and the listener, with his hearing aid turned on, checks a similar list. When

all 25 have been read, the result is checked with the reader's list. A hearing aid with a score of 70 per cent should give good service; an exceptionally good hearing aid will register 90 to 100 per cent.

c. Reading test. A third test that can be quickly and easily applied is a reading test. This impresses me as the most accurate of the three. All are weak[48] insofar as the voice intensities cannot be kept constant. My secretary reads from any book or article in a quiet voice. She is to pay no attention to me. Wearing the hearing aid to be tested, and turned on at its optimum high, I move away, or toward her, until I discover the greatest distance at which I can hear the details of her speech, paying especial attention to consonants. This distance gives the accomplishment factor sought.

CARBON AND VACUUM AID RESPONSE.

	Unaided Ear	Carbon Aid (Old Model)	Vacuum Aid (1939 Model)
Numbers	10 inches	11 feet	25 feet
Syllables (West Test)		74%	92%
Reading	8 inches	3 feet	22 feet

d. The value of these tests. The applicability of these tests and the contrasting efficiency of a few types recently available will be of interest. Vacuum aids were generously contributed for this review by three co-operating companies.[13, 49, 50] They had been adjusted to my audiogram before shipment. Though varying at certain points, they gave very similar responses in the quiet office, and are here contrasted with the responses for the carbon aid and the unaided ear. The better ear was used and the test was given in a quiet conversational voice.

This was in an office noise environment of 10-15 dcb., and the vacuum aids gave a much better response. Then I tried them in a noisy environment (general conversation, lecture, church, after-dinner speech). With the entire electric power turned on, the background noises and static from overloading were now so loud as to mask out the speaker's voice. Then the power was adjusted through the rheostat control to a point where the background noises no longer interfered. With one instrument, the current had to be turned down until but one-quarter of the power was available, making this instru-

ment much less efficient than my carbon aid. The second instrument did better but static still troubled. This illustrates the need for expert laboratory review at this point. Skilled adjustment would probably obviate this troublesome static. The third instrument was efficient with the rheostat turned down but one-third, making the net result considerably better than my carbon aid. This was very satisfying and justified the claim of the lack of distortion and greater amplification in the vacuum aid. But it illustrated the hazard of relying on a quiet test in an office when determining the suitability of a hearing aid.

e. Corroborative test. A rough corroborative trial for these tests was made by Elsie Staples in the Boston Guild for the Hard-of-Hearing on a small, unselected group of nine, each using his own hearing aid. Ages ranged from 25 to 60 years. There were two with otosclerosis, one with conduction, two with nerve, and two with mixed deafness, two not given. The percentage loss ranged from 40 to 70. Four used an Acousticon, three the Western Electric, and two the Radio-Ear. Six used air conduction earpieces, and three used bone. The increase in distance heard varied from three times at the minimum in a nerve deafness case to 28 times at the maximum in a conduction deafness case. The average distance increase was 10 times.

The West efficiency test was also used. This showed variations in hearing aid response of 44 to 76 per cent, with an average efficiency of 63 per cent. These results would indicate that some such simple office tests, though inaccurate, can be used with profit by the otologist to check the efficiency response of the hearing aid under consideration. Experience will tell him their relative value.

7. *Summary:* In advising a hearing aid for our patient, we first assure ourselves that it can do no harm. After establishing the need and persuading him to seek this help, we furnish him with his audiogram and direct him how to proceed. When he has selected the instrument that seems to suit him best, we insist that he take it home and see how it serves him in his daily environment. Before the final decision is made, the otologist strives to discover, in the light of his wider experience, whether the hearing aid performance is

satisfactory. This he determines by the patient's own story and by simple tests at the office. Some evaluate the hearing aid through a direct hookup with the audiometer receiver. Others use a calibrated voice transmission attachment. The problem is a complex one. The careful program of research outlined by the Committee on Standards, made up of acoustical engineers, gives promise of careful consideration and ultimate solution.

In the meantime, the otologist may employ simple tests consisting of numbers, nonsense syllables, and reading out loud, all carried out under as controlled conditions as time and circumstance will permit. In this evaluation, due regard must be taken to the masking effect that noises may have when amplified through the hearing aid under consideration.

V—CONCLUSION.

In closing, permit me to stress the great acoustic gain and personal comfort that accrues from a properly fitted hearing aid. I deem it the otologist's duty, as well as his privilege, to help his patient surmount his hesitation in seeking this help. He is in a strategic position to relieve him of his worry, and to guide him wisely. But, in order to really help him, the otologist must so inform himself that he can speak with authority. Familiarity has taught many the effectiveness of the carbon granule type of hearing aid. I have tried to review the salient advantages of this type and compare them with those of the modern vacuum aid. A recently developed midget vacuum tube is responsible for the rapid development of the latter type. Competition is keen. Several new concerns are joining the old-line manufacturers in presenting these models. I sense that, though its mechanical attributes and limitations are well known by the acoustical experts, the variety and multiplicity of these hearing aids, their lack of a common standard, and the inability of many servicing agents to scientifically adjust the amplification to the patient's need, will all result in mistakes and disappointments. The otologist can do much to remedy this through careful advice and kindly help.

Acoustic engineers are making new discoveries and adaptations. There has been remarkable progress. The making and

fitting of hearing aids is becoming an exact science. As one of the many who are hard-of-hearing, I bespeak the earnest support and continuing encouragement of this Society and of its members.

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DISCUSSION.

HARVEY FLETCHER, PH.D. (New York): I certainly think we owe Dr. Berry a debt of gratitude for spending so much time collecting the opinions from such a wide variety of sources and integrating them into this paper, together with the contributions he has made from his own experience.

There are one or two things which I should like to discuss, maybe with slightly different emphasis than Dr. Berry gave. Some of the statements are difficult to prove one way or another, and necessarily must be somewhat in the category of opinion. First, the question of carbon sets versus vacuum tube sets. I should like to point out that there is nothing inherent in a carbon microphone hearing aid which would make it have more peaks or more irregularity in its response than a vacuum tube set. I am sure Dr. Berry is correct, however, in saying that most carbon microphone hearing aids do have a more irregular response. That is not due to the carbon. That is due to the way the diaphragm actuates the carbon; it is not at all inherent in a carbon microphone.

With reference to the historical aspects of vacuum tube hearing aids, I am sure some of you remember that vacuum tube hearing aids were developed at Bell Telephone Laboratories over 20 years ago. The first sets that we made available were of the vacuum tube type, and some of them were portable, but we found that their weight and size and general inconvenience more than outweighed their superior quality.

Now, as time has gone on, as Dr. Berry has pointed out, some of these sets are beginning to be quite portable and are being reduced to somewhere near the same size as carbon sets; however, I do not think we should be misled by the fact that they are called vacuum tube sets; some of these vacuum tube sets are no better in response than the carbon microphone sets and will give no better results. It all depends on the kind of carbon microphone set and the kind of vacuum tube set in question. It is true that there are some vacuum tube sets with better response than some of the carbon microphone sets. Moreover, the use of vacuum tubes has made possible sets having a greater volume range than was available in carbon microphone sets. I wish to emphasize, however, that the mere use of vacuum tubes in a hearing aid does not assure a superior performance.

If I had to guess what the future would be, it would be a different guess from that of Dr. Berry. My guess is that we are going to have more carbon microphone sets ultimately, because carbon microphones and amplifiers using carbon elements can be designed to produce excellent hearing aids and, inherently, they have in them the possibility of making much smaller sets and more convenient sets. I do not think anybody can prove that. I am simply stating that as my guess. Before that can be accomplished, however, some very fundamental research work must be done on the carbon itself. That research is going on for other reasons, and the hearing aid field will no doubt profit from it when the work is completed.

With reference to the question of bone conduction versus air conduction, the problem which you have heard discussed from time to time, I presented a short discussion to this Society on that subject some time ago. At the present time, the best bone conduction receivers are less efficient than air conduction receivers in the sound volume which they send into the inner ear by about 40 dcb. Therefore, if a normal hearing person listens with a bone conduction receiver, the sounds are weaker by 40 dcb. compared to those produced by the best air conduction receivers. Possibly in another five or 10 years we may have that differentiation reduced down to 30 dcb or less; right now it is 40. A few years ago, it was 50. Then the bone conduction receiver had very much less use. If, as at present, the differentiation is about 40 dcb., then for the bone conduction receiver to yield better results than the air conduction receiver, the difference in the hearing of the patient by bone

conduction and air conduction must be more than 40 dcb., provided the same electrical power is delivered to each receiver. This differentiation between the receivers would not hold if unlimited power were available, such as in some vacuum tube hearing aids operated from house power mains. Then a bone conduction receiver could be used for any type of hearing lesion because it would be possible to vibrate the whole head through amplitudes as large as necessary. If the head is vibrated sufficiently, the impulses must reach the nerves, if there are any nerves left, and produce the sensation of hearing. The only limitation to the use of a bone conduction receiver under these circumstances would be one of physical discomfort produced by the excessive vibration of the receiver.

As the art now stands, I would hesitate to agree with Dr. Berry that patients in the hearing loss range from 60 to 80 dcb. prefer the bone conduction receiver. I should say the reverse; that the air conduction receiver would be more likely to be used. I do not believe it is possible for a hearing loss to be of the order of 80 dcb. unless there is considerable involvement of the nerves. Under such conditions it is unlikely that the air conduction hearing loss will be more than 40 dcb. greater than the bone conduction loss.

There is one other question that I should like to discuss; namely, the suitability of different tests for determining the effectiveness of hearing aids. I think you will have most success by using sentences. If numbers or sentences are used for test material, and I prefer sentences, your patients will differentiate very clearly between different types of hearing aids. The line of demarcation between where the patient understands practically everything and where he understands practically nothing is very sharp.

Not very much material is necessary to obtain a significant result with sentences; usually 20 sentences are sufficient. On the other hand, if words are used as test material it may be necessary to use 200 or 300 in order to obtain the same result.

Material has been presented on this subject before and I am sure you will find it in the literature.

Dr. Horace Newhart (Minneapolis): We have listened with great interest and profit to two men pre-eminently qualified to speak on this subject which is one of rapidly growing interest to the otologist.

It has become an important function of the otologist to help his aurally handicapped patients obtain hearing devices which are best adapted to their individual requirements. We should bear in mind that these patients frequently are financially as well as aurally handicapped, and the purchase of a hearing aid represents a capital investment; also that those having a severe handicap are the most difficult to fit satisfactorily. It should be noted that no two persons present identically the same hearing problem, and no two instruments yield identical performance characteristics.

With the many improvements in their construction and performance and the successful production of wearable vacuum tube devices, the problem of prescribing and selecting a hearing aid has become more complex and, to the patient, more confusing. The number of persons who can be benefited by a modern hearing aid and who need expert help in securing one has been materially increased. More intense competition between distributors further complicates the problem.

Thus, the interested otologist who is qualified and adequately equipped to render this special service can save his patient time and help him to avoid the disappointment of purchasing an instrument which is not satisfactory, discrediting both the one who, without investigation, recommends a given device and the manufacturer.

With the information obtained by a thorough otological examination and familiarity with hearing aids which have been pronounced "acceptable" by

the Council on Physical Therapy of the American Medical Association, he can safely recommend or prescribe an aid of the type and make which will yield the best results. For this service he should charge the patient a reasonable fee, and never accept from the distributor any commission, bonus or fee in violation of recognized principles of medical ethics.

He will be able to frankly tell his patient whether his present condition warrants the purchase of a hearing device; if he can successfully use a carbon microphone aid, or will require one of the newer vacuum tube devices which give greater amplification and fidelity.

The patient should be allowed the privilege of a trial period on a reasonable rental basis that he may determine which of two or more aids of apparently equal merit will yield the best results when subjected to the ultimate test; that is, the articulation test, with which every otologist should be familiar.

This procedure is not favorably regarded by the average salesman, who is primarily interested in selling the aid which affords him a livelihood; however, with some encouragement from the otologist, the salesman often sees the wisdom of conceding this privilege.

The representatives of the leading manufacturers whose products have proved "acceptable" to the Council of Physical Therapy are carefully selected and trained, instructed to observe the rules of ethical salesmanship and to avoid making diagnoses or give medical advice. They render excellent service in supplying modern hearing aids, assembled by selective amplification, whereby the performance characteristics of the assembled instrument within certain limitations, meets the user's requirements as indicated by his audiogram.

Unfortunately for the otologist, the agent is so well equipped that the patient often gains the impression that he knows more about his otological condition than the otologist. Unless the latter informs himself adequately concerning the elementary principles underlying the subject of hearing aids and is equipped with the needed physical apparatus to insure the best results, he will be at a disadvantage. He will do well to co-operate understandingly with the ethical vendor of "accepted" hearing aids.

It is suggested that the otologist, in referring a hard-of-hearing patient to the hearing aid representative, impress upon the former the fact that the purchase of a hearing device will not prove the complete solution of his hearing problems. The patient should be advised of the importance of making every effort to conserve his residual hearing by keeping in close contact with an interested, understanding physician or otologist to guide him in maintaining his general health and in correcting any known conditions which may result in further hearing deficiency.

It is also the function of the otologist and the general practitioner to warn their hard-of-hearing patients against exploitation by unscrupulous sellers of inferior or worthless hearing devices. Such racketeers travel about the country, especially in rural areas, selling their wares for cash at whatever prices they can obtain and speedily departing for parts unknown before they can be apprehended. A note of warning should be sounded regarding possible unpleasant litigation from real or alleged injury resulting from unskillful attempts to obtain a cast for making an individually fitted plastic earpiece for the midget receivers now available. An unfortunate case of this kind has recently come to our attention.

The manufacturers of hearing aids are rendering a most valuable service by constantly improving their products. Many whose instruments are of real merit have not yet submitted their wares to the Council on Physical Therapy for acceptance.

The otologist would do well to encourage all ethical manufacturers of meritorius hearing aids to submit their products to the Council in order that the

consuming public, otologists and the makers themselves may be protected from inferior products.

Dr. Kenneth M. Day (Pittsburgh): Dr. Berry referred to me in speaking about the possible injurious effect of the hearing aid. I will frankly state that I am developing what I believe to be an occupational type of deafness, largely my own fault, due to overuse of my hearing aid. During the past five years I have been developing an increasing loss of hearing for high tones. The margin of leeway that I have between the point of intelligibility and the threshold of pain is not very great. I try to hear too much, and if extraneous noises are very loud they do cause pain. I believe that this problem of occupational deafness from the hearing aid is probably applicable only to those cases with a fixation of the stapes as they have no buffer or brake against loud, jarring noises. The injurious effect would be increased if there is much "peaking" in the instrument.

One comment I would like to make. For the past five years I have been wearing an instrument wired with a receiver to each ear, and I am sure that, with less amplification, it increases intelligibility by at least 30 per cent. This is due to the fact that sound impulses are transmitted through both auditory nerves. I can understand and differentiate conflicting sounds when using both ears, whereas with one ear, though the sounds seem louder, they blur together and I cannot differentiate them; however, plugging both ears necessitates a complete re-education of one's own voice. It is very uncomfortable to have both ears plugged, and there are very few people who will submit to the discomfort and become re-educated to the point where they are willing to wear a receiver in each ear.

As to the relative value of the air conductor and bone conductor, I believe a great many people are wearing bone conductors who should be using air conduction. Although I have about 4-to-1 bone conduction in the lower ranges, I can hear better with air conduction because of a loss of bone conduction for the higher tones. When there is a marked loss of hearing by bone conduction in the range between 1,024 an 4,096, the air conduction instrument should give better hearing. Patients will often deny this because of lack of psychologic adjustment to their infirmity. They will swear until they are black in the face that they hear better with bone conduction because they have to plug an ear if they wear air conductors; that means distortion of their own voices, discomfort and a feeling of being conspicuous.

Dr. Edmund P. Fowler (New York): I just want to make one point, and that is, did you notice (excuse my being personal, but I have to be in this case) how Dr. Berry and Dr. Day spoke, how clear their voices were and how well modulated? They wear hearing aids, and the hearing aid is a great help in enabling hard-of-hearing people to properly modulate their voices. It trains them to speak at a proper loudness when it is turned on, and even when it is not turned on it is not usually given any credit for this effect.

Dr. Berry: I thank the speakers. There is quite a bit in the paper I wasn't able to give.

THE ENDAURAL ROUTE.*

DR. D. E. STAUNTON WISHART, Toronto.

The demonstration that the difficult operation of fenestration for otosclerosis can be performed through the external auditory meatus has aroused many otologists on both sides of the Atlantic to perform the simple and radical mastoid operations by this route. In past years this route has been condemned as not in keeping with the principles of sound surgery — but now there are voices which proclaim it the route of the future. It is, therefore, fitting that the merits and demerits of the route should be thoroughly discussed.

The endaural approach to the mastoid is made through a triangular window in the posterior wall of the external meatus. This triangle is difficult to define in words and in practice. On the accuracy with which it is incised depends the adequate exposure of the field of operation, and the avoidance of damage to the cartilage of the auricle. An appreciation of the anatomy is essential and is afforded by study of three illustrations from Spalteholz, which have been slightly modified.

On the external surface of the ear — see Fig. 1 (Fig. 876 — of the right ear, viewed from without) — the landmarks of importance are the circular end of the external meatus, the tuberculum supratragicum, the crus helicis and the incisura auris anterior.

The crus helicis is a slightly curved elevation immediately above and behind the circular outer extremity of the external meatus, and is the lower extremity of the helix. The tuberculum supratragicum is the upper extremity of the tragus. Between the crus helicis and the tuberculum supratragicum is a small furrow.

Fig. 2 (Fig. 879 — right ear cartilage, viewed from without) shows that beneath this furrow there is no cartilage, for it shows that the tragus is formed by the lamina tragi

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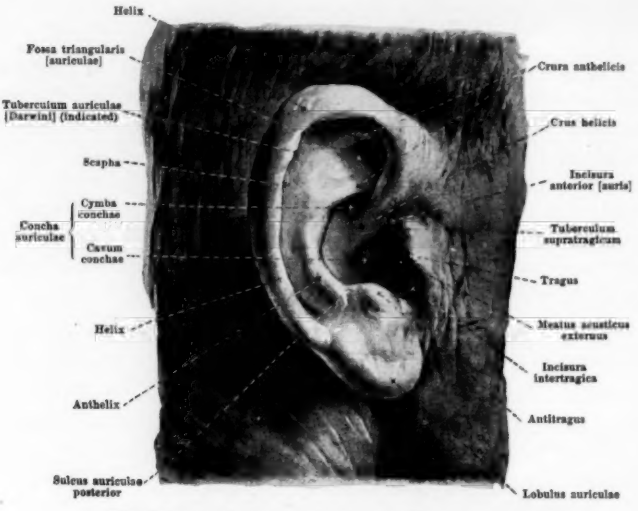
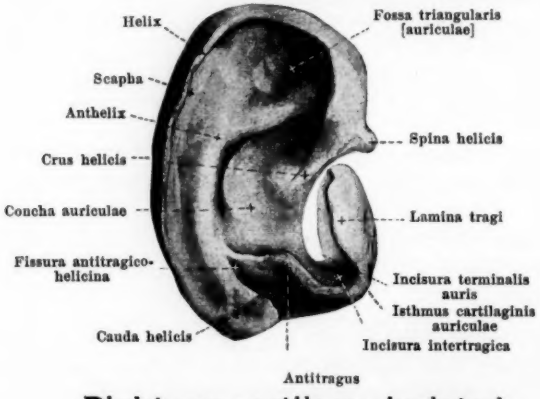
and that this lamina is separated by a space from the crus helicis. This space is the incisura auris anterior.



Right auricle, viewed from without (drawn from life).

Fig. 1. (Fig. 876, p. 800, Spalteholz. Hand Atlas of Human Anatomy, Vol. III.)

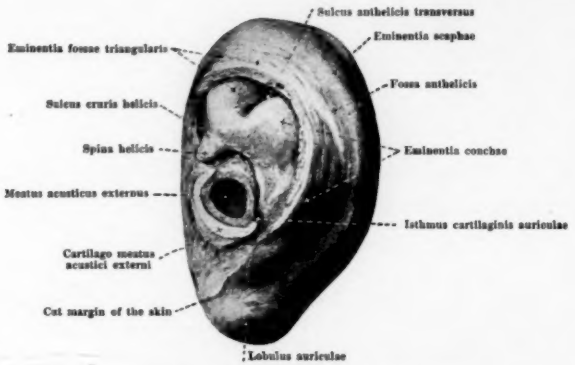


Right ear cartilage, isolated,
viewed from without.

Fig. 2. (Fig. 879, p. 801, Spalteholz. Hand Atlas of Human Anatomy, Vol. III.)

Figs. 2 (Fig. 879) and 3 (Fig. 877 — right auricle, viewed from within) show that the upper and posterior half of the cartilaginous meatus is devoid of cartilage. It is this portion of the external meatus which is removed in the formation of the triangular window of the intrameatal approach to the mastoid. When this window is properly fashioned, no cartilage is included in the excised tissue, and perichondritis and deformity become unlikely complications.

The accurate delineation of this triangle is rendered difficult by the mobility of the pinna and of the external meatus. It



Right auricle, viewed from within.
(The auricle has been removed from the head by a clean cut.)

Fig. 3. (Fig. 877, p. 800, Spalteholz. Hand Atlas of Human Anatomy, Vol. III.)

should be outlined by three scratches — vertical, horizontal and oblique (see Fig. 4).

The vertical scratch is the easiest to visualize and is, therefore, described first. It is a curved line commencing at 7:00 o'clock in the outer rim of the external meatus and curves backward and upward inside the rim to about 12:00 o'clock, where it leaves the rim and proceeds upward in the incisura auris anterior, as far as the operator desires. As can be seen from a study of Fig. 2, it should hug the anterior edge of the cartilage of the auricle without cutting it.

The horizontal scratch commences at 12:00 o'clock in the roof of the external meatus, at the junction of the cartilag-

inous and bony portions and runs horizontally outward to reach the upper end of the vertical scratch.

The oblique scratch runs from the internal end of the horizontal scratch downward, backward and outward to reach the lower end of the vertical scratch (see Fig. 5).

When the operator is satisfied with the scratch outline, incisions should be made through these scratches to the bone and the triangle of soft tissue removed (see Fig. 6). Lempert advises that the oblique scratch be incised first, then the horizontal, and finally the vertical.

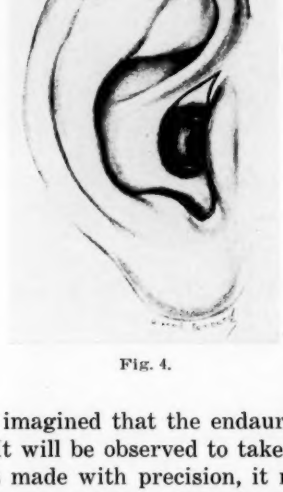


Fig. 4.

It must not be imagined that the endaural incision is performed quickly. It will be observed to take about 10 minutes as a rule. Unless made with precision, it may jeopardize or even ruin the subsequent course of the fenestration operation. The deliberateness, thoroughness and slowness with which an ear surgeon performs this approach is usually directly proportional to the number of times he has used the route.

Figs. 4, 5 and 6 are reproduced from Lempert and show the window that is cut in the posterior part of the membranous portion of the external meatus. This window can be made larger by making a fourth incision, commencing where the first and third cuts join and curving upwards along the

attachment of the helix on the side of the head as far as desired.

It must not be thought that too much detail has been given regarding the incisions. Experience will show the immense amount of truth packed into the terse recommendation that these incisions be made "with the greatest accuracy and precision." A few days will repair the ill effects of maceration from improper retraction but not those of inaccurate or ragged incisions.

The softness of the tissues makes the triangle difficult to delineate — but hemorrhage makes the incisions still more

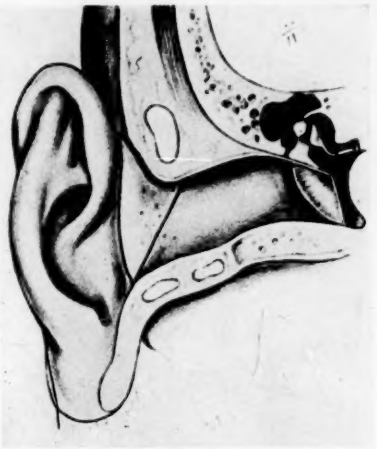


Fig. 5.

precarious. The fenestration operation is performed under local anesthesia where hemorrhage is a minimum. But general anesthesia is the method of choice for the majority of simple and radical mastoid operations, and in spite of the impregnation of the tissues with adrenalin, hemorrhage at times will render the making of the incision awkward.

With a periosteal elevator, the periosteum is separated from the whole of the outer surface of the mastoid bone; that is, downward to the tip, backward to the posterior border of the mastoid or beyond that if necessary — far upward on the squama — and far forward on the root of the zygoma.

The illustrations demonstrate that by the use of retractors any part of the outer surface of the mastoid can be exposed, but it must not be imagined that exposure can be made of all the area of the mastoid at one time (see Fig. 7). When the zygomatic region is exposed adequately by heavy retraction on the anterior retractor, there will be almost no exposure of the mastoid region because the mastoid retractor will be held loosely (see Fig. 8). Similarly, when the postsinus region is being explored there will be marked retraction on the posterior retractor and almost none upon the anterior retractor (see Fig. 9). The hardest region to expose adequately is the

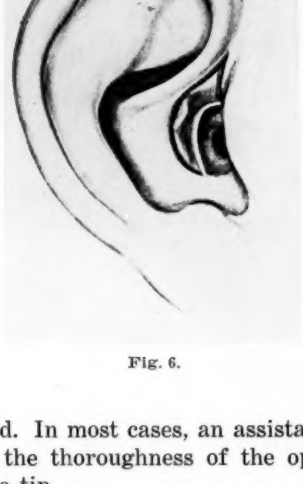


Fig. 6.

tip of the mastoid. In most cases, an assistant or observer is unable to judge the thoroughness of the operative work in the bottom of the tip.

The exposure depends on the size of the window and on the mobility of the auricle and the soft tissues. The higher the incision is extended upwards in a curved direction the greater the exposure. Such an extended incision, however, cuts into the fibres of the temporal muscle. What is involved by this will be mentioned later. When the superficial tissues are normal, as in the type of patient recommended for the fenestration operation for otosclerosis, and as will be the case in some

of the patients on whom a radical mastoid or even a simple mastoid operation is performed, the exposure is in every way adequate. When the pinna and the superficial tissues are stiffened and thickened by inflammatory reaction, as is frequently the case in acute mastoiditis, the mobility of the tissues is very greatly impaired. When the postaural reaction is intense and the tissues are grossly thickened, it is with considerable difficulty that a large mastoid is satisfactorily operated upon through the ordinary postaural incision;

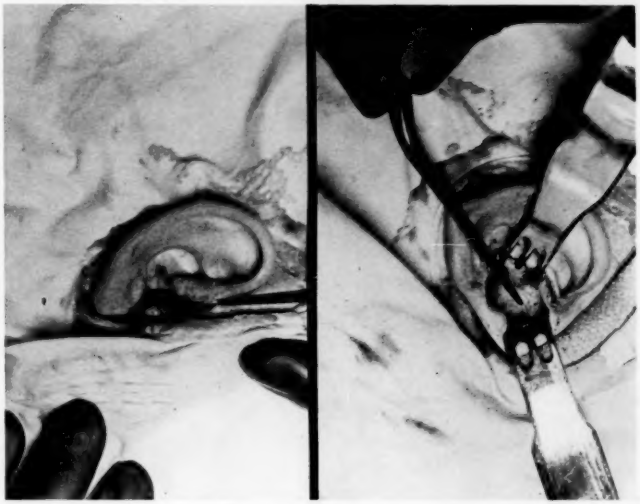


Fig. 7.

under such conditions the endaural route becomes almost an impossibility.

In order to achieve adequate exposure, the incision may have been continued up along the edge of the helix. It may go far enough to divide the lower border of the temporal muscle and this could produce infection of an uninfected region. The writer sutured this incision in one case in an attempt to minimize the final scar but was rewarded by an abscess. Since then whenever he has had to unduly extend the incision upward he has bathed it in tincture of metaphen and at the conclusion has packed the incision wide open with iodoform

gauze. In these cases he has had no postoperative infection and, in spite of the packing, the skin edges have subsequently approximated so that the final scar probably will be invisible.

The retraction of the soft tissues is of obvious importance. The difficulty involved is less apparent, but is indelibly impressed on every observer of the fenestration operation and, therefore, must be stressed here. It involves heavy dragging on one retractor and next to none on the other. Hence, no mechanical contrivance will do the work. It must be done

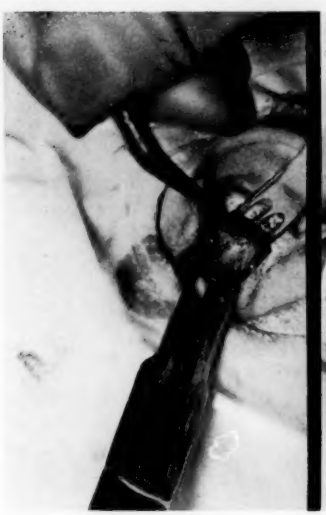


Fig. 8.

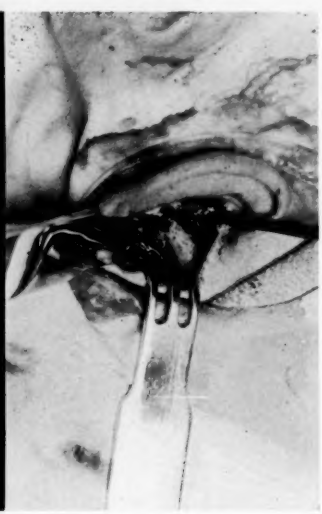


Fig. 9.

by hand, and in such a way as not to move the patient's head, not to interfere with the operator, and not to macerate the retracted tissues. Tremendous traction is at times required. And as the "opening time" is lengthened by the endaural route, an assistant is required who is conscientious, very strong and very patient. The retraction of the tissues is no enviable task.

Great damage can be done to the edges of the incisions unless the retractors be peculiarly well held. Lempert is fortunate in always having the same retractor holder. The average otologist will be required to put up with ever-chang-

ing assistants — some of them indifferent, or worse. The writer was surprised to find how rapidly the macerated tissues recovered. The reader can see this for himself by study of Figs. 10, 11, 12 and 13.

The fenestration operation requires a two-man team — one to hold the retractors and one to operate. (The usual assistant is not wanted. A surgeon as skilled in the fenestration operation as the operator can act as assistant — but no one less competent should be allowed.) The simple and radical oper-

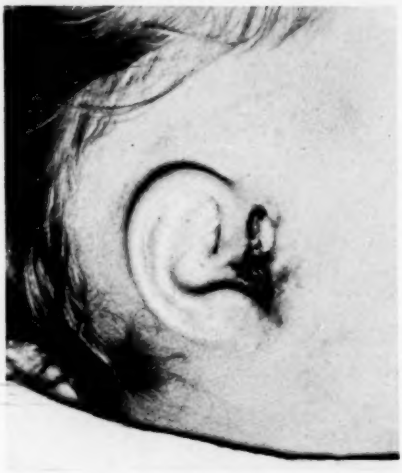


Fig. 10.

ations by the endaural route require a three-man team, the above two and an assistant to sponge.

The writer estimates that his "operating time" is lengthened considerably by the endaural method. But this is a guess, for the "operating time" varies with the conditions found. As experience in the endaural method is gained the "operating time" will undoubtedly be reduced but, because the incision demands such care, the retraction of the parts is so difficult and the assistance is rendered more awkwardly, he believes that the "operating time" will always be longer than by the postaural route, even though there is time gained by not having to suture.

At the conclusion of the simple mastoid operation by this route, dry, fluffy gauze is applied to the auricle and bandaged on.

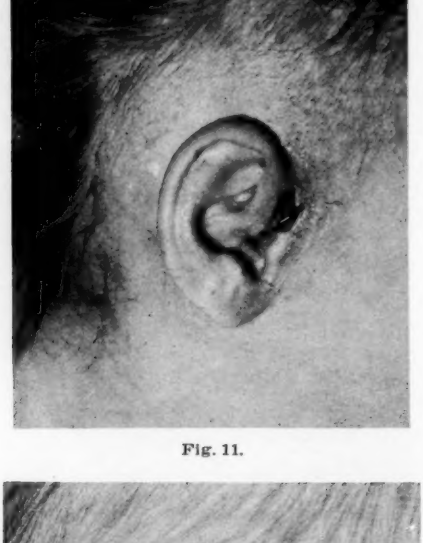


Fig. 11.

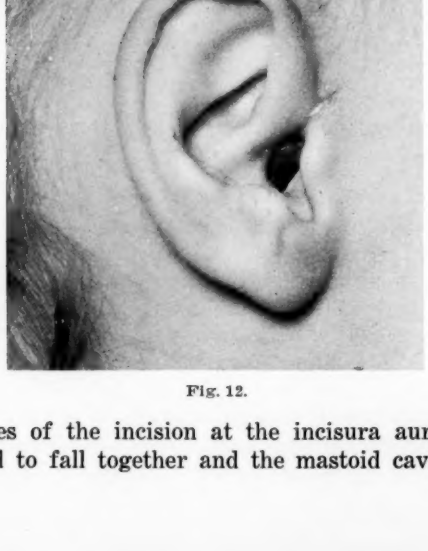


Fig. 12.

The edges of the incision at the incisura auris anterior are allowed to fall together and the mastoid cavity may or

may not be loosely packed, as the operator desires. When the mastoid excavation is small, there is no indication for either drainage or packing. The gauze is changed one or more times

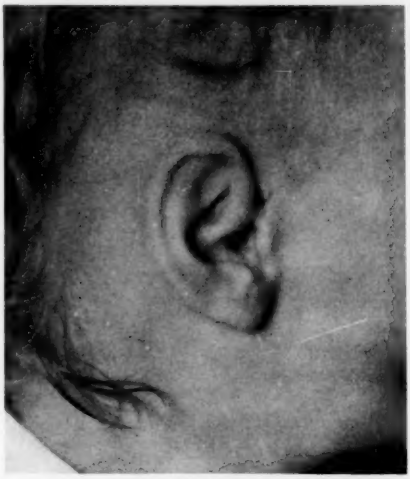


Fig. 13.

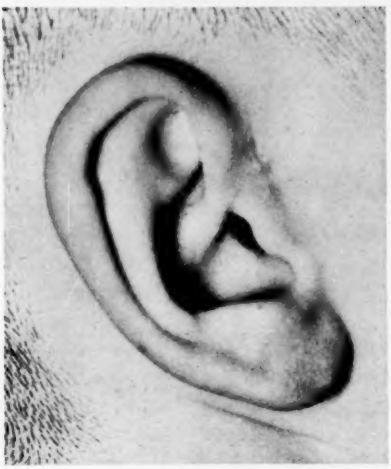


Fig. 14.

daily, depending on the speed with which it is saturated with discharge; at each dressing the external canal is thoroughly dried. When the excavation is large or there is a deep

postsinus pocket or a deep zygomatic exenteration, loose packing or a drainage tube is required. The essential principle is that the mastoid excavation must granulate from the depth toward the opening in the skin and, hence, the intrameatal sinus must be kept open. The external meatus is extraordinarily sensitive and, in consequence, the postoperative care of these cases by a nurse or house surgeon is either excessively feared or inadequately performed, or both.

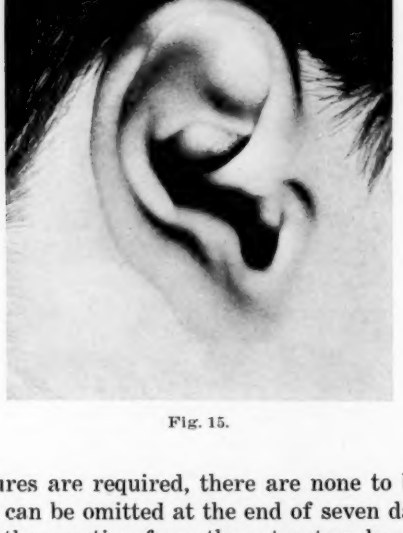


Fig. 15.

As no sutures are required, there are none to be removed. The bandage can be omitted at the end of seven days, because by that time the reaction from the retractors has nearly subsided and the supra-auricular skin edges have approximated. Vaseline to the latter, and loose gauze over the meatus, held on by a "triangle," becomes the routine dressing; at the same time the skin surfaces of concha and canal are cleaned. This postoperative attention is easy but requires a skilled hand, for a nurse cannot see the wound and cannot understand what she is doing — and the patient greatly fears the dressing, for the canal is excessively sensitive.

For the purpose of investigating the endaural approach, a number of children were operated upon at the Hospital for Sick Children, Toronto, and subsequently as many as could

be collected for the purpose were shown to some interested otolaryngologists. Only the 11 cases seen by these specialists

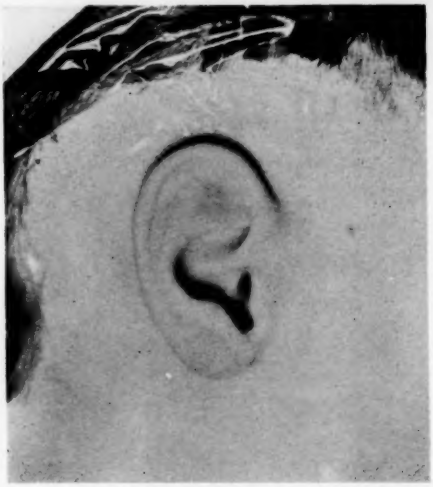


Fig. 16.

are here reported (see Table I). The remainder are not known to have any visible or undesirable result.

TABLE I.

	Age	Acute or Chronic O.M.	Simple or Radical Operation	Days in Hospital	Seen After Operation	Constriction of Canal	Special Notes
1.	5	Acute	S.	17	13 mos.	0	Sinus had to be reopened.
2.	5	Acute	S.	16	12½ mos.	0	Excessive granulations
3.	13	Chronic	R.	14	12 mos.	0	
4.	5	Acute	S.	10	11½ mos.	0	
5.	9	Acute	S. Bil.	36	9½ mos.	0	One intra-meatal; one post-aural.
6.	9	Acute	S.	20	8½ mos.	0	Lateral sinus very far forward.
7.	6	Acute	S. Bil.	33	7 mos.	?	Nearly closed at operation; abscess temporal region.
8.	11	Chronic	S.	20	5½ mos.	0	Healed, dry and open 6½ months after operation.
9.	12	Chronic	R.	19	2½ mos.	0	
10.	7	Acute	S.	26	2 weeks	0	Unusuallylargepostsinus abscess.
11.	4	Acute	S.	25	7 days	0	Very small child; small meatus.

In the first nine cases the writer handicapped himself unnecessarily by carrying the incision as short a distance as possible upward. This greatly lengthened the operating time but, as can be seen from the four illustrations (see Figs. 14, 15, 16 and 17), which are typical, the postoperative appearance of all was excellent.

In the tenth case, the operation was greatly facilitated by extending the incision upward. Because many of the readers of this paper may require to follow this procedure, sev-

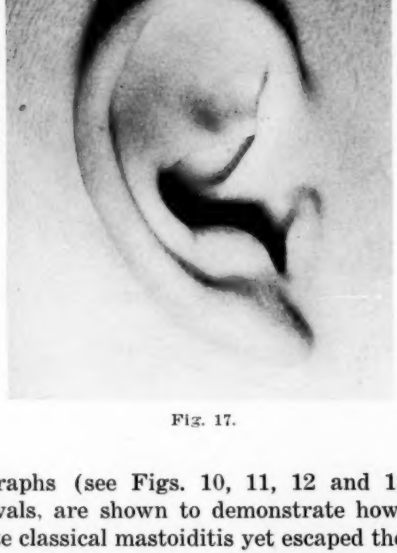


Fig. 17.

eral photographs (see Figs. 10, 11, 12 and 13), taken at stated intervals, are shown to demonstrate how this patient with an acute classical mastoiditis yet escaped the feared complication of infection of the temporal tissues. In this the patient and operator must be considered fortunate. This case is further remarkable because it had a very large cellular mastoid with as great involvement behind the posterior border of the lateral sinus as I ever saw in a patient so young. There is no doubt that every corner of this postsinus region was adequately seen and exenterated. None of my patients developed keloid. Two had a radical mastoid operation, and one a simple mastoid operation on ears that had been dis-

charging for many years and whose hearing was almost *nil.* The latter (Case 11) when examined five and one-half months after operation was continuing to discharge both from the middle ear and from the intrameatal sinus but is now completely and satisfactorily healed. The remaining patients all had acute mastoiditis — and in some the postaural or zygomatic edema was excessive. Two cases had had a simple mastoid operation performed on the opposite ear, and comparison of the appearance of the two sides drew comments unanimously in favor of the intrameatal approach.

Operation was performed on the child, age 4 years, to demonstrate that even at that age the operation is feasible. The lateral sinus was found extremely far forward in the sixth case. The seventh patient had pre-operative involvement of the posterior canal wall, and fear was expressed that stenosis of the meatus would result: for some weeks there was 50 per cent narrowing; but at the time all the children were inspected one observer thought the canal was stenosed one-fifth, while the remainder failed to see any stenosis or thought it negligible. In nearly all, some difficulty was found in keeping the intrameatal sinus open. In two, it was found closed too soon and bulged, but in each case it was opened quickly without anesthetic and gave no further trouble. It had been feared that complications might arise from the fact that via the endaural route the drainage does not take place from the most dependent part. None arose, possibly because considerable care was taken in the postoperative treatment, so that this objection, while sound theoretically, has not been found of practical importance.

It can be seen from the table that the postoperative stay in hospital was on the average at least twice as long as is usual for mastoid patients that have the postaural approach. In some patients the endaural opening drained freely and remained clean; such patients were quickly out of hospital. In other patients, however, postoperative granulations became excessive and their wounds were, consequently, very difficult to clean. Such patients dreaded the daily dressing. The latter could not be done by a nurse but had to be done by myself when I could spend the time, or by a relatively inexperienced house surgeon, and the stay of such patients in hospital was excessive.

In spite of this longer confinement in hospital, the care in the out-patient department of the hospital lasted much longer than for those patients who have the postaural approach. The reason for this is that the endaural sinus cannot be properly seen by dressing nurses or house surgeons and, hence, they have no adequate appreciation of the condition of the wound. The postoperative care of his intrameatal cases is an individual task for the one who operated, just as is the care of the postoperative radical cavity; but, unlike the latter, it requires daily and not intermittent attention. At a busy hospital an operating surgeon must delegate the postoperative care of his patients to others; to proceed thus with intrameatal cases would be to court disaster.

The writer has had postoperative narrowing of the canal in only one case. As described above, that narrowing has almost disappeared and is negligible. All the surgeons who have seen my endaural operations or who examined the patients after operation have looked carefuly for it; they seem to expect it. I can testify that it need not occur.

The parents of the children operated upon and most of the nurses and some of the surgeons who saw the above series of patients are enthusiastic about the procedure. Were their comments to be our sole criterion, judgment would be in favor of the intrameatal route.

It must not be inferred, however, that the postoperative appearance will always be an improvement on the postaural method. The writer has seen a postoperative appearance which was anything but pleasant. The circular external orifice of the meatus had been converted into a much larger, pear-shaped opening, with the extension upward in front of the helix. Keloid was present in some of the scars I have seen. These objectionable features have occurred in the practice of a surgeon whose technique is excellent and are, therefore, the more worthy of record as a warning, lest the hearers and readers of this paper imagine that the postoperative appearance will always be better than that produced by the postaural incision.

The writer has also seen recently in consultation a case on whom the intrameatal simple mastoid operation had been performed on both ears. The patient had not been well since

operation many weeks previously. X-ray and subsequent operation by the postaural route revealed that neither mastoidectomy had been adequately performed.

In spite of what has been said, and in spite of the illustrations, there will be many otologists who will doubt the possibility of the complete eradication of all diseased tissue; for any ear surgeon of extended experience is aware of the embarrassing variations in the internal geography of the mastoid bone, and in the pathological conditions met with and knows there are great advantages in having the entire field of operation completely visible by the surgeon and by his assistant also. To these I would recommend attending a Lempert otosclerosis operation. They will be amazed at the exposure that is achieved; they will be able to confirm my statement that when the superficial tissues are normal the tissues can be so moved that an assistant can view and assist in clearing all areas except the base of the tip and a cellular postsinus region.

To see these two regions, the surgeon requires unusually good illumination and he requires the luck not to be embarrassed by hemorrhage. But the more abnormal the superficial tissues the greater the difficulty of inspecting the depths of the cavity and the less the help of a watchful assistant, fragments of dead bone may escape detection, and bits of undermined, undernourished cortex may be allowed to remain — so that the objection of these doubters is valid. The simple and radical mastoid operations are performed for the safe removal of all diseased tissue. Whether this has been achieved or not is apparent to an observer in all cases where the postaural approach is used. If the surgeon operates by the endaural route, there will be plenty of instances where the operating surgeon alone can know whether he has met this essential surgical requirement.

It is reasonable, however, to state that, granted good illumination and sufficient experience with the route, a competent ear surgeon *can* perform all simple and radical mastoid operations by the endaural route. This statement must be qualified by the admissions that there will be cases where the operation will be performed awkwardly and slowly, and that when the case turns out to be hemorrhagic, operation by the end-

aural route will not be performed without danger to the patient.

In short, the difficulties of safe, complete eradication of all disease in the mastoid via the endaural route are such as to prevent its use by any ear surgeon except one of unusual experience and one who has given himself special training in its use.

It is obvious that the writer has unearthed many disadvantages for the intrameatal route, but before enumerating these attention should be given to the merits of the method, which are:

1. The avoidance of a postaural scar.

2. The better visualization of the middle ear, particularly the post-tympanic area.

3. The plastic work required in the radical operation is done in advance.

4. A mechanical advantage in drilling the window in the horizontal canal and in drilling the front wall of the attic.

The first of these would seem a reason unworthy of a surgeon's consideration; but, on the other hand, the appeal of this to the public is so great that to pass by this argument without comment is to play into the hands of the exploiter-type of surgeon. The modern mastoid scar is close to the postaural crease, is completely hidden by the auricle, in the majority of cases is so thin and small that it can be found only with difficulty, and it causes no disfigurement. When the postaural scar after operation by an experienced surgeon is objectionable in appearance it is proof that the operation disclosed a condition requiring wide open exposure and unusually long drainage; even such is hidden by the lobe of the ear. It has been recorded, however, that operation by the endaural route may produce a disfiguring scar or keloid; and disfigurement in this case has nothing to hide it. The first supposed advantage of the endaural route is, consequently, not an advantage.

2. Adequate view of the middle ear is not required in the simple mastoid operation but is of prime importance in the radical operation and in the fenestration operation for oto-

sclerosis. The postaural incision permits adequate exposure of and approach to all parts of the middle ear except the post-tympanic space. This area, the cleaning of which is of vital importance in the radical operation, cannot be seen through the postaural approach unless the incision be carried in circular fashion upward and forward so that the pinna and external canal can be pulled so far forward that the operator may look directly down the external meatus. This postaural approach affords the necessary view and the necessary operating space only when the soft tissues are violently displaced. The endaural approach does allow the operator a full view of the posterior half of the margin of the tympanum and, therefore, is of great assistance in the radical mastoid operation.

3. The use of the postaural incision in the radical operation inadequately meets sound surgical principles, in that a second aperture has to be made through the external cutaneous surface; namely, a window has to be made through the tissues of the posterior wall of the meatus into the newly fashioned cavity. The violation of principles lies in making a second incision through the external tissues when one might have sufficed. The endaural route in the radical operation, on the contrary, requires only one opening on the skin surface and in that respect is superior to the postaural route.

The endaural incision, as described above, removes a portion of the meatal soft tissue that is ordinarily displaced towards the mastoid bone at the termination of the radical mastoid operation. The endaural route does, however, allow the best possible utilization of the remainder of the posterior part of the external meatus which is reflected over the facial ridge on to the floor of the antrum. There is no doubt that the skin lining the bony meatus can be beautifully dealt with by the endaural route. It can be thinned as much as desired and reflected over the facial ridge and then adequately held against the bone until it takes. By a slight modification of the skin incisions and of the subsequent technique, the skin described as excised and thrown away can be utilized to line the mastoid bowl. The result is that by dint of adequate packing the lining of the mastoid and antrum portions of the cavity by skin is greatly facilitated. As a result of this, epithelization of the radical excavation takes place quickly and well.

The endaural route, therefore, offers certain distinct advantages in the radical mastoid operation.

4. The drilling of the window in the horizontal canal is the most difficult part of the amazingly difficult technique of the fenestration operation for otosclerosis. Anyone who has tried the procedure on the cadaver knows that this drilling is much more easily carried out through the endaural route than it is through the postaural route with the auricle held forcibly forward. Similarly, the drilling of the front wall of the attic is better performed when the long axis of the drill is in the line of the external canal. These mechanical advantages are so great that, once experienced, a surgeon has no doubts about them.

In contrast to these advantages, the text has shown that in the author's hands the endaural route has the following disadvantages in comparison with the postaural route:

1. The incision is difficult; unless it is performed with precision, the result may be jeopardized.

2. An extra assistant is required for the unusually difficult job of holding the retractors.

3. There is a danger of introducing infection in the temporal region.

4. The operation takes a longer time.

5. There is inadequate exposure of the tip and postsinus regions.

6. Damage by the retractors could give rise to perichondritis of the auricle.

7. The wound after operation is much more sensitive than the postaural wound.

8. The postoperative attention cannot be safely detailed to the usual out-patient dressing clinic; *i.e.*, it gives the surgeon more personal work to do.

9. The postoperative healing period is lengthened.

10. Excessive granulations are treated with difficulty.

11. Secondary mastoiditis requires the attention of a surgeon who understands the intrameatal route.

12. Constriction of the external meatus is a theoretical possibility.

13. Objectionable scars or fissure at the top of the canal entrance have been known to occur and are conspicuous.

14. Unusual illumination is required.

15. It is a difficult approach to learn, so that the ordinary operator cannot be expected to learn it properly.

The writer has no quarrel with the use of the endaural route for the fenestration operation for otosclerosis. He believes, further, that those who desire to perfect their technique for that operation must use that route in practice upon suitable patients requiring simple and radical mastoid procedures.

The fact that these two procedures can be performed by the endaural route does not prove, however, that the endaural route is the method of choice for either of these procedures.

The simple and radical mastoid operations call for the removal of infected and diseased tissue — the fenestration operation for otosclerosis does not. The simple mastoid excavation should be made to heal from the depth outward. The radical and fenestration excavations are intended to remain cavities. When the surgical requirements of operations are the same, the surgical principles governing the operations must remain the same, and vice versa.

Whiting long ago laid down sound principles for the modern mastoid operation, *viz.*, the "complete eradication of all diseased bone, in the shortest time commensurate with thoroughness, by methods designed to reduce to a minimum the dangers incident to the operation and calculated as well to induce speedy and permanent healing." These principles still hold.

The writer believes he has proved that, while these principles can be followed by the endaural method to some extent, that method does not follow them as completely as the standard postaural route. He concludes that the endaural route is not the method of choice for the simple and radical mastoid operations.

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170 St. George Street.

DISCUSSION.

DR. JAMES A. BABBITT (Philadelphia): There can be but one opinion about Dr. Wishart's careful paper in discussion of "The Endaural Route," and that is complimentary.

With great precision he has outlined entrance procedure, discussing carefully its difficulties and dangers, and emphasizing the importance of accuracy, for adequate exposure of the field and avoidance of cartilaginous danger. His discussion of this approach represents study and is valuable.

Through this and other parts of the paper we find a most tolerant attitude toward the insistence of Dr. Lempert upon exact technique. Dr. Wishart's discussion is fearless and original, and while we may not entirely agree with it, we must respect it.

We are quite in agreement with the necessity for great care in incision, the advisability of the postauricular operation in acute mastoiditis, and the necessarily personal attention of the operating surgeon in postoperative dressings, as well as the advisability of having any secondary mastoiditis which may arise operated by one who understands the endaural route.

In the cases viewed by the writer, we have not observed the danger of introducing infection laterally to the temporal fascia; that it took an essentially longer time than the postauricular route; nor that it has produced a secondary perichondritis.

The sensitivity of the wound and the period of postoperative healing varies so with the conditions surrounding the actual operation that it is difficult to evaluate it. If the periosteum is well lowered, there would seem to be an adequate exposure of the tip and postsinus regions.

In one phase we are hardly in agreement — and that is the limited attention given to postoperative granulations and constrictions. (We must allude to the magnificent record of those cases portrayed on the screen.)

Much of this is doubtless due to carelessness in injuring the posterior canal wall, but, even so, these granulations, even to atresia of the canal, are very disturbing, indeed, and may require a long attention, and perhaps contribute much to the sensitivity of the wound. The complaint of patients about this long-continued and disturbing care of the granulations has been emphasized.

In conclusion, I would repeat that more emphasis should be placed upon granulation and atresia and less upon the difficulties in procedure, secondary inflammatory disturbance and lengthening of the postoperative healing.

Beside excluding the simple mastoidectomy from the endaural route, we should be in favor of further limiting radical mastoidectomy to those cases in which there is intricate or precisional work required, such as fistulization or reorganization in the middle ear, and direct vision is important. It seems rather doubtful that the endaural approach will be popularized except in these cases.

The general impression of this paper is that of an honest attempt at fairness, though it does rather condemn the endaural route. The advantages of the latter have been fairly given but we feel that its disadvantages might be conditioned by the variable technical operative ability of those attempting this procedure. This, however, is a fine paper — the best we have had of this sort — and the author is to be heartily commended.

Dr. D. E. S. Wishart (Toronto): I thank Dr. Babbitt for his very kind remarks. He considers that the danger of infection in the temporal region need not be stressed. When you read my paper, you will find from the table that I had only one infection in the temporal region. Still, with all respect to Dr. Babbitt, I do think that I was fortunate.

I am in complete agreement with the rest of his remarks. His criticism is correct that I have not sufficiently emphasized the treatment of postoperative granulations. They are of great practical importance.

ANATOMY OF THE CRANIAL BLOOD SINUSES WITH PARTICULAR REFERENCE TO THE LATERAL.*†

DR. BARNES WOODHALL, Durham, N. C.

The anatomy of the lateral sinus and its contiguous structures has been a subject of study for many years and has been presented in detail upon many occasions. Although the descriptive anatomy of any portion of the human body may be a static matter, disease of any structure is essentially dynamic in character. In a consideration of the anatomy of the cranial venous sinuses, it is important then to review the subject in the light of our changing conception of disease processes in which these major efferent channels of the brain play a part.

Since the two lateral sinuses are the major venous end-channels of the brain, it is obviously of importance to understand in the first place what anatomic variations may appear in their structure and thus influence their function as drainage channels. A knowledge of such variations is valuable in the surgical treatment of otitic sinus thrombosis, certain tumors of the posterior cranial fossa, neoplastic or inflammatory lesions of the neck and in the procedure of jugular ligation for any reason, in order to anticipate or appreciate cerebral symptoms of venous stasis. As a corollary, variation of lateral sinus structure must be recognized in order to interpret the significance of the Tobey-Ayer test as a test for venous sinus thrombosis. In the second place, the relation of the lateral sinus, the jugular bulb and their tributary vessels to the ear are of primary importance to the otologist in his consideration, study and operative attack upon otitic infection. No matter what particular anatomic information may be sought, it is most important to seek a means of translating our empirical knowledge gained at the dissecting or autopsy table to a definite adjunct in clinical diagnosis. In this paper an effort is made to delineate the anatomic vari-

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†From the Department of Surgery, Duke University School of Medicine and Hospital, Durham, N. C.

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ations of surgical import in the region of the torcular herophili and involving the lateral sinuses and to illustrate the significance of such variations in the venous sinus pattern by appropriate clinical cases.

Gross Anatomy: Classical descriptions of the anatomy of the venous sinuses of the dura mater are available in any textbook of anatomy, varying but little in detail. It is important to realize what cerebral areas are drained by each sinus and the extracerebral circulation that is available in case of interference with the chief pathways.

The longitudinal sinus occupies the attached margin of the falx cerebri. Commencing at the foramen cecum, it runs from before backward, passing beneath the region of the anterior fontanelle and the sagittal suture to the occipital protuberance, where its main channel divides to one side or the other and is continued as the corresponding lateral sinus. Its inner surface presents the openings of the superior cerebral veins, more numerous at the caudal part of the sinus and partially concealed by fibrous bands. The longitudinal sinus receives the superior cerebral veins, united in four main trunks, a frontal, a precentral, a postcentral and an occipital. These veins drain the superior and mesial portions of the cerebral hemispheres. The precentral and postcentral trunks anastomose with the middle cerebral and Sylvian veins, which are in turn tributaries of the cavernous and superior petrosal or lateral sinuses. The capacity of these anastomosing vessels is variable, and the longitudinal sinus remains the chief venous channel for the drainage of the cortical surface indicated.

The inferior sagittal sinus occupies the free surface of the falx cerebri, runs from before backward, and receives tributaries from the falx and the mesial aspects of the cerebral cortex.

The straight sinus is situated at the junction of the falx cerebri and the tentorium cerebelli and is a continuation of the inferior sagittal sinus. It receives the superior cerebellar veins and the great vein of Galen, the latter being the only venous channel of any consequence draining the nuclei in the region of the third and lateral ventricles and much of the white matter of the hemispheres. The straight sinus runs downward and backward to empty its blood in most cases into the lateral sinus of the side opposite that into which the main channel of the longitudnal sinus is continued. It communicates with the confluence of the sinuses in this region.

The lateral sinuses begin at the occipital protuberance and pass laterally and forward in the attached margin of the tentorium to the base of the petrous portion of the temporal bone. Each then leaves the tentorium and passes downward to reach the jugular foramen. The important tributary veins are those of the diploe, the mastoid emissary veins and the condyloid emissary veins. They also receive the superior and inferior petrosal sinuses.

There are three principal and many accessory communications: 1. The plexus of the foramen magnum, the uppermost of the spinal plexuses, anastomoses with the transverse occipital sinus in front and the posterior occipital sinuses behind. Further, these plexuses, or their trunks, the vertebral and posterior jugular veins, anastomose with the posterior condyloid emissary vein and the mastoid emissary vein. 2. The ophthalmic vein, entering the cavernous sinus, and thus the petrosal sinuses, anastomoses with the facial and temporal veins. 3. The middle meningeal veins form a communication between the longitudinal sinus and the pterygoid plexus. The accessory communications are innumerable because the origins of the external jugular vein and of the facial portion of the internal jugular vein communicate with those

of the intracranial veins practically everywhere over the vault and the base.

Embryology: Any abnormality that may develop and attain permanency in the adult venous structure has its anlage in the embryological state. The detailed studies of Streeter[1] clearly elucidate in what localities one may expect such variations in the sinus system and the reasons for their occurrence. In view of their importance in understanding the abnormal, they are quoted at some length.

"In the primary type of circulation the arrangement for the drainage of the capillaries of the head consists bilaterally of one main channel, the 'primary head vein,' that starts in the region of the midbrain, runs caudalward alongside of the brain-tube and terminates at the duct of Cuvier. The primary head vein is composite in origin. That portion of it rostral to the vagus nerve is an intrinsic vein of the head; the remaining caudal portion is in reality a neck-vein and constitutes the anterior caudal vein — eventually the internal jugular vein. Together these portions form a continuous channel, the primary head vein, into which the blood from the capillary sheet immediately investing the brain-tube is drained by means of anastomosing venous loops. These loops are arranged more or less in the form of three plexuses — the anterior dural plexus, the middle dural plexus and the posterior dural plexus. Other small tributaries empty into the primary head vein, thereby draining the structures ventral and lateral to the brain-tube, such as the nerve ganglion masses and the maxillary and mandibular gill-bars. A large one comes from the eye region and eventually is modified into the ophthalmic vein.

"From this simple group of drainage channels are eventually derived all the adult venous sinuses. The metamorphosis which they undergo is based on a series of circulatory adjustments that are made necessary by certain changes in their environment, the two most conspicuous being the changes in the region of the cartilaginous capsule of the labyrinth and the still greater changes involved in the growth and marked alteration in the form of the brain. Among the factors involved in these circulatory adjustments may be mentioned the reduction of plexuses into simple channels, the conversion of channels into plexuses, the total obliteration of established channels and the change in position of channels. Under this latter phenomenon, there is to be recognized a 'passive migration' where there is a change in the position of the vein wall itself, due to the movement of its environment, which exerts a flexion or traction force upon it. We also recognize a 'spontaneous migration' where there is a change in position of the blood stream only, where in a circumfluent manner the blood stream develops a new channel in the adjacent loops of the plexus, with a corresponding dwindling of the previously used channel. The 'replacement channel' might be mentioned as another type of spontaneous migration, in which the venous channels are changed in position and direction in this process of adjustment. In the replacement channel there is the formation of a new channel and the obliteration of an old one, as in other types of spontaneous migration. It, however, differs from them in that it is not a gradual and progressive change in position, but an abrupt and immediately complete one. Furthermore, the new channel lacks the morphological characteristics of the old one. With these various factors in mind, one can readily follow the steps by which the primary head vein and its tributaries gradually merge into adult dural sinuses.

"While the three head plexuses are spreading upward, the outlines of the dura mater and the arachnoid spaces make their appearance, and first of all in the ventral parts. This results in a general separation or cleavage of the more superficial primary head vein and its three tributary plexuses from the subjacent vessels that arise from and drain the capillary sheet directly investing the brain-tube. This deeper system,

however, continues to drain into the former at certain places, notably in the more dorsal parts. The primary head vein and its three tributary plexuses thus become established as a true dural system, as distinguished from the deeper 'cerebral veins' belonging to the arachnoid-pial membrane. The diploic veins are a later subdivision of the dural system. The superficial veins of the head belonging to the integument and soft parts are separated off in the more ventral regions and from there spread upward over the head independently of the dural system. We then have for the head three separate systems: 1. the superficial layer belonging to the integument and soft parts; 2. the middle layer belonging to the dura and the diploe; 3. the deep layer of the cerebral vessels belonging to the brain. It is the middle layer, or dural system, that is exclusively concerned in the formation of the dural sinuses and whose changes in form and position we are now following.

"In the region of the cartilaginous capsule of the labyrinth adaptive changes in the dural channels occur early. Owing to the marked elaboration of these structures in this region, the course of the primary head vein, ventrolateral to the otic capsule, becomes an unfavorable one. If it persisted it would be tortuous and remote from the area drained; instead, this part of it becomes obliterated, and during this obliterating process an adjustment is made in two ways: 1. a channel is established in the venous plexus above the otic capsule, and through this the middle dural plexus thereafter drains caudally into the loops of the posterior dural plexus; 2. the anterior dural plexus, which originally drained into the primary head vein, completely reverses its direction of flow and drains through anastomosing loops into the middle dural plexus and through the newly established channel dorsal to the otic capsule.

"In this way a complete trunk for the drainage of the head becomes established, which is everywhere dorsal to the primary head vein as far as the jugular foramen, where it is continuous with the internal jugular vein. Of the primary head vein there is left, in addition to the cardinal portion of it, or internal jugular vein, only that part in the region of the trigeminal nerve. This may now be spoken of as the 'cavernous sinus.' Into it drains a vein from the base of the brain and the veins from the orbital and maxillary regions; whereas it, in turn, drains upward through the original trunk of the middle plexus, which is now the superior petrosal sinus, into the newly established dorsal channel . . . this dorsal channel is the transverse sinus, of which that part between the superior petrosal sinus and the jugular foramen forms its sigmoid portion. Thus, in the 21 mm. embryo the dural channels in the region of the temporal bone have acquired essentially all their permanent connections, with the exception of the inferior petrosal sinus, which appears a little later. Otherwise there remains to complete the adult condition only a certain amount of passive migration in accommodation to the changes in the adjacent parts.

"The adjustment in the dural channels rendered necessary by the protracted growth of the hemispheres extends much later in fetal life. A large part of this adjustment is accomplished by spontaneous migration of the principal channels, and for this reason a venous plexus is essential. We thus find in the neighborhood of the advancing occipital pole of the hemispheres a continuous persistence of the transitory or embryonic dural plexus from which are evolved all the veins of the falx cerebri and of the tentorium cerebelli.

"An anterior subdivision of the plexus extends forward in the median line as the plexus sagittalis, being interposed as a vertical curtain between the hemispheres. Among its dorsal meshes is developed an asymmetrical longitudinal channel which we know as the 'superior sagittal sinus.' In its early stages this channel is made up of several collateral anastomosing veins. The eventual single channel is formed in the anterior portions by the selection and enlargement of the most

favorable vein with a corresponding disappearance of the others. In the posterior portions there is apparently some coalescence of adjacent veins. The anterior part of the sinus is completed first. As the hemispheres extend backward, the sinus correspondingly elongates itself by incorporating the more caudal loops of the plexus. Transverse sections through this portion of the sinus in older fetuses thus usually reveal incomplete coalescence of the separate loops. The sagittal plexus very early exhibits a tendency to drain more to one side of the head than to the other and usually toward the right side. As the superior sagittal sinus becomes established we thus find that caudalward it is usually continuous with the ventral main channel of the right anterior plexus (or tentorial plexus, as it is better called in the late stages), which eventually forms a part of the right transverse sinus. The straight sinus is formed in the ventral part of the sagittal plexus and its caudal adjustment is essentially like that of the superior sagittal sinus. It may drain chiefly toward the right or left plexus or equally toward both.

"In embryos between 35 and 50 mm. long, we can recognize a main channel of the tentorial plexus that is to become the transverse sinus. If we disregard the sigmoid portion of it, it forms a fairly straight line with the internal jugular vein. In the interval between the 50 mm. embryo and the adult, the transverse sinus bends backward until it comes to lie at an angle of 90° with the internal jugular. This marked change in position is accomplished in large part by spontaneous migration, by the repeated shifting back of the main blood current into more caudal loops of the plexus, with subsequent dwindling of the discarded anterior loops. As the sinus becomes more definitely established, the tentorial plexus becomes relatively smaller and the final change in position is completed by passive migration; that is, actual traction on the vein wall by its environment. In this change in position of the transverse sinus the superior sagittal sinus and the straight sinus participate and we find in the adult, at the point where they meet, an anastomosis, the confluens sinuum, which is usually plexiform in character and represents the last trace of the embryonic tentorial plexus."

It is evident, then, that there are several localities where development may fail or where, through coalescence or shifting of venous channels, variations in the adult may ensue. In the first place, the development of the embryonic venous channels is bilateral and a connection must be made along the longitudinal sinus and at the confluens sinuum. Secondly, the anterior vascular plexus, or the tentorial plexus, as it might be called in the later stage of development formed after the obliteration of the primary head vein, may not persist, either wholly or in part, resulting thereby in the absence or attenuation of the lateral sinus of that side. Finally, because of constant elaboration and change during migration, local alterations in the final sinus form may persist.

Just why the right lateral sinus should carry the greater volume of the venous flow is still a moot point. The longitudinal sinus carries more blood than does the straight sinus; hence it follows that whichever lateral sinus receives the blood from the former will be the larger. Therefore, all the

theories presented have sought to explain why this sinus veers to the right at the torcular herophili.

Von Meyer[2] attacked this problem as early as 1877. He found no evidence in the cranium for the right-sided predominance, but based his reasoning upon the anatomical arrangement of the cervical venous vessels. He noted that the right-sided venous outflow from the base of the brain to the heart was short and straight, whereas that on the left, including the left innominate vein, was longer and twice bent in a gentle curve. In addition, the thoracic respiratory movements were more easily able to exert their emptying influence upon the straight right side. Because of these two facts, he believed that the right side would tend to develop at the expense of the left. His theory made no effort to explain the percentage of cases, approximately 12 to 15 per cent in large series, in which the left lateral sinus is predominant.

In 1904, Smith[3] published his observation that the occipital lobes of the brain were asymmetrically developed, the left extending further posteriorly than the right, due probably to the greater development of the left visual centre. To the superior volume of the hemisphere in this region he ascribed the turning of the longitudinal sinus to the right. On the other hand, he postulated that the lesser bulk of the right occipital pole favors the venous return on this side, and this might lead to the greater development of the right transverse sinus. In the few cases in which the right visual centre was larger, Smith stated that the left transverse sinus was over-developed. To support this ingenious theory, Smith studied anthropoid apes and found that in the majority of animals the brain showed no localized over-development and the venous pattern was symmetrical.

Some years later, Bluntschli[4] sought to explain this constant asymmetry from the standpoint of comparative anatomy. He stated that in the lower apes the sinuses were more clearly defined and more regular in distribution than in man and in the anthropoid apes. This was especially true in the region of the torcular. He found in most cases that the longitudinal sinus divided at this region and was more or less evenly distributed to the two lateral sinuses. As the higher forms of animal life were studied, he noted that the angle

between the median plane and the lateral sinus increased; that is, the lateral sinus changed from an oblique, lateral and upward course to a more and more transverse direction. This change in angle of outflow, Bluntschli attributed to the greater development of the cerebrum and the gradual retreat of the cerebellum beneath the occipital lobes. With this change in direction he encountered greater sinus asymmetry and greater right-sided predominance.

He also felt that the suppression of the left superior vena cava in the higher animals played a part in sinus asymmetry. As the animal orders ascend, the left innominate vein and the left superior vena cava gradually become connected with the right superior vena cava and the latter vessel enters the heart alone. He believed that this unfavorable path, occurring coincidentally with the shift in position of the lateral sinus, influenced the development of right-sided predominance.

Zeiger[5] pointed out several obvious criticisms of the above theories. He felt that the physical factors influencing blood flow in the large vessels of the neck were far too complex and were too poorly expounded at the time of his writing to justify one in using the meagre evidence of longer and more tortuous pathways on the left side to explain this asymmetry. He adduced the anatomical evidence that in animals with a left superior vena cava, sinus asymmetry is still found. On the other hand, in horses, donkeys, hogs and cats, in which the outgoing venous channels are quite roundabout, an asymmetry is found. In the third place, he did not believe that one can adequately measure one-half of the brain volume and didactically say that one-half of a cerebral hemisphere is larger than the other.

Zeiger brought up the influence of "handedness" upon the over-development of one side of the brain and thus perhaps upon sinus asymmetry. Right- or left-handedness may be observed primarily in the arm and secondarily influence the contralateral growth of the hemisphere. One arm may be longer than the other and may be used to greater advantage; definite handedness may develop therefrom. Zeiger pointed out that the percentage of left-handedness with a longer left arm is about 10 per cent, and that arms of equal length are

found in about 12 per cent of individuals. The total of 22 per cent compares favorably with the number of variations in the venous sinuses from the common pattern of right-sided predominance. He stated, from the comparative anatomical point of view, that only in the higher apes is there an appreciable degree of right- or left-handedness as shown by differences in the length of the bones of the arms, and only in the higher forms does one meet an appreciable percentage of sinus variation. Zeiger wrote that in man, asymmetry of the sinuses does not occur until after birth, concomitant with greater asymmetry of the bones of the arms.

It is doubtful whether the final sinus form is attained in the human at birth. The sinus system of the newborn is more complex than in the adult; the occipital sinus, for instance, reaches a tremendous size as compared with the adult form. Streeter, however, stated that in 18 20-mm. embryos studied, 16 showed a clear-cut tendency at this early stage toward right-sided predominance. One is forced to the unsatisfactory assumption that this major and primary variation in sinus pattern is part of phylogenetic development. It is due

TABLE I.

Direction of Venous Outflow from the Brain as Noted in Studies by Various Authors.

Cases	Author	Date	Right	Left
Skull —				
	Hunauld[6]	1730	—	0
14	Morgagni[7]	1741	—	0
100	Rudinger, N.[8]	1876	71	27
512	Sperino, G.[9]	1884	269	78
100	Von Spee[10]	1896	68%	13%
200	Le Double[11]	1903	137	29
400	Sturmhofel, O. A.[12]	1903	232	61
	Zeiger[5]	1923	60-70%	0
Jugular Foramen —				
126	Theile, F. W.[13]	1855	46	24
100	Rudinger[8]	1876	71	27
449	Korner, O.[14]	1889	264	109
530	Budde, K.[15]	1891	40-64%	26-30%
	Zuckerkandl, E.[16]	1892	54-60%	25-30%
Dural Preparations —				
50	Dumont, J. N.[17]	1894	75%	—
35	Henrici, C., and Kikuchi, J.[18]	1903	43%	—
42	Mannu, A.[19]	1907	66%	—
60	Vernieuwe[20]	1920	43%	—
	Zeiger, K.[5]	1923	66-68%	—
35	Pfeiffer, A.[21]	1930	60%	%
50	Edwards, E. A.[22]	1931	24	21
24	Gibbs, E. L., and Gibbs, F. A.[23]	1934	—	—

perhaps to differences in outflow channels, perhaps to intracranial asymmetry that has been in turn influenced by asymmetrical extremity growth.

During the course of a study of the clinical picture of venous back-pressure phenomena in the brain as a result of lateral sinus thrombosis, 100 dural preparations of this region were studied *in situ*, and later dissected or injected with contrast media. The sinus patterns of the torcular and its contiguous sinuses fell into four well defined groups, irrespective of occipital sinus abnormalities which appeared among the four main groups, and these types may be well illustrated by reference to clinical cases involving thrombosis of the sinus system.

1. Common Pool Type: This is the classic and textbook picture of conformation of the sinuses and is actually encountered far less often than are the other patterns noted here. In this variation the longitudinal and the straight sinus meet in a more or less common pool, from which the blood is directed to the lateral sinuses. The latter are ordinarily equal but may vary in size within narrow limits. Nine instances of this simple type of conformation of the sinus were observed in this series, a number that closely agrees with that given in previous reports. In all but two, the lateral sinuses were equal in size; in these two, the right lateral sinus was slightly larger. A very small occipital sinus entered the pool in three instances; a moderate-sized sinus entered the pool in one. In two a small occipital sinus entered the left lateral sinus within 1 cm. of the pool, and in three no occipital sinus was present. The markings of the skull were practical equal.

CLINICAL ILLUSTRATION–I.*

A female white infant with an acute onset of fever, vomiting, generalized convulsions and spasticity, progressing to coma and exitus within five days. Thrombosis of torcular herophili with retrograde straight sinus thrombosis in "common pool" sinus pattern. Hemorrhagic infarction of basal ganglia.

*The cases presented in this paper were studied while the author was Resident in Surgery, The Johns Hopkins Hospital, Baltimore., and he wishes to extend to Dr. Dean Lewis, then Professor of Surgery, his gratitude for Dr. Lewis' aid and encouragement.

P. H., a white female, age 13 months, H. L. H. 95,988, was admitted with a history of vomiting and fever for the preceding three days. She was the only child of normal parents. Delivery and postnatal life were normal until the onset of her present illness. She had had no infectious disease with the exception of one mild upper respiratory infection. Three months prior to admission, diphtheria toxoid had been given.

Three days before admission, the baby vomited in a projectile manner and spent a restless night crying and whimpering. The following morning the child was quite drowsy but took her feedings normally. She was taken to an outside physician, who made a diagnosis of tonsillitis and predicted further attacks of vomiting. Her temperature was 100° per rectum. That evening the baby vomited three times and her mother felt that the baby did not recognize her. The child rested quietly except for continuous movements of the right arm and leg. The arm was flexed, the leg extended, and both were described as undergoing coarse, regular, jerking movements. The child whimpered all night. On the morning of admission, the child was comatose and did not take her feedings. Jerking movements of the right extremities continued. A public health nurse obtained a temperature of 103° per rectum and referred the child for hospitalization.

Upon admission, the temperature was 103.4° per rectum, pulse 160, respirations 54 and blood pressure 130/80. The child was well nourished but dehydrated. She lay in a stuporous condition. Her right arm and left leg were flexed; the other extremities extended. At short intervals, perhaps 30 to 60 seconds apart, the right upper and lower extremities would abruptly develop convulsive movements, those of the leg being gross and flail-like, those of the arm being clonic movements of short excursion. These seizures lasted 15 to 30 seconds.

Respirations were most irregular and appeared to be both Cheyne-Stokes and Biot in character. At times shallow breathing gradually increased to respiratory movements of wide excursion, receding then to periods of apnea. At other times, rapid deep breathing was immediately superseded by apneic phases.

The mental state was almost that of coma. To extremely painful stimuli there was a withdrawal response. To tactile

stimulation there was no response. There was no response to light flashed into the eyes. To loud auditory stimuli, only a flutter of the eyelid was elicited.

On the ward, it was noted that she lay with the head turned to the right, with deviation of the eyes to the same side. At frequent short intervals the right upper and lower extremities would shake in clonic movements of short excursion, the right forearm being flexed, the fingers tightly clenched. In contrast to the lack of involvement noted in the dispensary, the left side now showed the same movements to a less degree. During these attacks the child would make chewing movements of slight amplitude. The trunk was not rigid during these attacks nor was the head retracted. At any time, however, upon lifting the shoulders the trunk would become rigidly extended on the thighs for about 10 seconds. There was a bilaterally positive Babinski response. The fontanelle was firm but not bulging. There were no other pertinent physical findings.

Preliminary laboratory studies were negative except for a leukocytosis of 12,000.

Lumbar puncture upon admission showed a xanthrochromic fluid under slight pressure, estimated but not measured. There was a 4+ Pandy reaction, with 830 R.B.C. and 120 W.B.C., 90 per cent of which were lymphocytes. The clinical impression was that of subarachnoid hemorrhage.

On the following day, all extremities were rigid and there were continuous twitching movements of the right side of the face. Lumbar subarachnoid drainage was done with withdrawal of 40 cc. of fluid under definitely increased pressure. The cellular elements were increased with 520 R.B.C. and 2,610 W.B.C. Smear and cultures of the fluid were negative. The child improved markedly following this procedure but four hours later became cyanotic, respirations became rapid and gasping, and exitus occurred.

Pathological Note: The entire brain was red and swollen, with a definite cerebellar pressure cone. The superior longitudinal sinus contained a reddish postmortem clot. Where the superior longitudinal sinus entered the lateral sinuses there was a grayish, friable clot. This extended into both lateral

sinuses for a distance of 1.5 cm. The straight sinus and the veins of Galen were distended with a reddish clot. Some of the veins on the dorsal surface of the brain contained homogenous red clots.

On section, the thalamus and caudate nuclei on each side, and the putamen on the left side, were softened and infiltrated with blood, and had the appearance of a hemorrhagic infarct. Anteriorly, the changes extended far anteriorly into the cau-

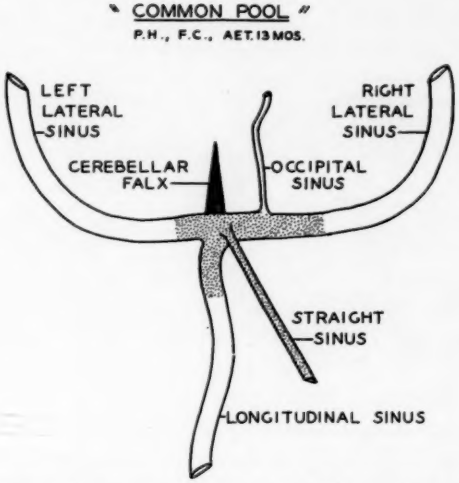

Fig. 1. Clinical illustration I. Thrombosis of torcular herophili, lateral sinuses and straight sinus in "common pool" sinus pattern.

date nucleus. The ventricular ependyma, anteriorly, was slightly blood stained.

The choroid plexuses in the lateral ventricles showed vessels distended with reddish gray thrombi. This change in the choroid plexus was especially conspicuous on the right side and extended into the plexus of the aqueduct. The septum lucidum and the columns of the fornix were softened and showed small hemorrhages. There were small hemorrhages, too, in the globus pallidus on the left side.

The necrosis and hemorrhage extended back into the pulvinar of the thalamus but did not appear to invade the cor-

pora quadrigemina. In the tail of the corpus callosum there were some small hemorrhages. Further sections showed that the choroid plexus in the posterior tip of the right lateral ventricle was hard and enlarged. Its vessels were distended with thrombi. The tissue immediately surrounding the ventricle was softened and there were small hemorrhages scattered through it. Far out in the white matter, the small vessels were all distended but the tissue was firm. The veins in the midbrain which drain the choroid plexuses and lie just beneath the tail of the corpus callosum were all distended with firm, red clots.

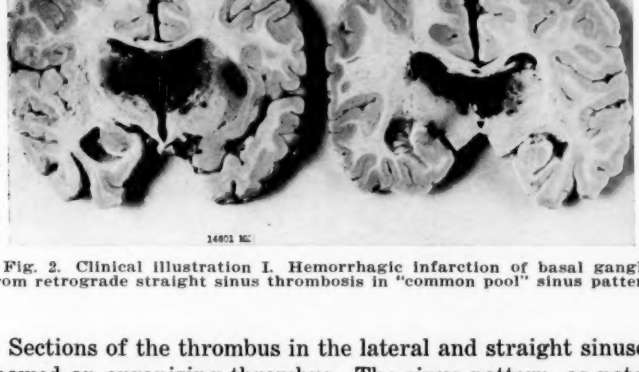

Fig. 2. Clinical illustration I. Hemorrhagic infarction of basal ganglia from retrograde straight sinus thrombosis in "common pool" sinus pattern.

Sections of the thrombus in the lateral and straight sinuses showed an organizing thrombus. The sinus pattern, as noted in the illustration, is that of the classical "common pool" type, a conformation that has been described in 9 per cent of anatomical preparations.

2. Plexiform Type: This is the type of variation of the sinuses that should be expected from the bilateral embryologic development of this region as detailed in the studies of Streeter. Many subtypes are possible, but there is usually adequate intercommunication between the lateral sinuses. The longitudinal and the straight sinus may each be divided into two divisions, equal or unequal, and the lateral sinuses may be equal in size, but usually are moderately dissimilar. The straight sinus may be divided into two branches, equal or

unequal, with the longitudinal sinus single, the latter flowing usually to the right with the smaller half of the straight sinus, or this may be reversed. The longitudinal sinus may be divided into two equal or unequal branches with the straight sinus single, the latter usually flowing to the left side with the smaller branch of the longitudinal sinus. There is generally more pronounced difference in the size of the lateral sinuses in these last two subtypes. There were 56 examples of this type of sinus pattern, and practically all the theoretical subtypes were observed.

The plexiform type of sinus pattern illustrates well the tendency toward right-sided predominance, with the larger volume of the longitudinal sinus directed toward the right, and the lesser volume of the straight sinus directed toward the left, or opposite side. In only one instance was there a wide disproportion between the volumes of the lateral sinuses, and in only 11 were there noticeable differences as great as two to one. There are uniformly full channels of cross-circulation at the torcular, through the divided sinuses or through a combination of these and accessory channels.

CLINICAL ILLUSTRATION—II.

A white male infant with congenital ureteral obstruction, admitted with opisthotonos, generalized spasticity and tonic convulsions. Straight sinus thrombosis in a plexiform sinus pattern demonstrated at autopsy. Hemorrhagic infarction of basal ganglia.

R. R., a white male infant, age 10 weeks, H. L. H. 86,531, was admitted for the treatment of persistent vomiting. The patient was the only child of healthy parents. His birth and development during the first eight weeks of life were quite normal. Two weeks before admission, he began to vomit and this persisted without remission. One week before admission, abdominal distention was observed, which likewise persisted.

Upon admission his temperature was 99.8°, pulse was 120 and respirations were 48. The blood pressure was recorded as 160/100. The baby was moderately dehydrated and was semicomatose. The skin was mottled and cyanotic. There was a smooth, well defined, kidney-shaped mass in the left flank, and a similar but less well defined mass in the right flank.

Palpation of each mass elicited a pain response. There were bilateral positive Babinski phenomena. The clinical impression was one of congenital malformation of both kidneys, with renal failure and hypertension.

Five days later, bilateral nephrostomy was performed. During the preoperative and postoperative periods, progressive bulging of the anterior fontanelle was observed. On the day after operation, edema was noted in both fundi. On this date, all four extremities and trunk became rigid and the child remained in a position of opisthotonos. Bilateral ventricular puncture demonstrated xanthochromic fluid with 2,300 R.B.C. and 3,300 W.B.C. Exitus occurred 74 hours after operation, immediately preceded by continuous tonic convulsions.

Pathological Note: The external appearance of the brain was practically normal. The specimen of dura showed part of the longitudinal sinus and both lateral sinuses. On sectioning the brain, around the ventricles, extending out into the white matter, particularly involving the greater part of the thalamus and extending forward to involve the basal nuclei, and forward and backward about each lateral ventricle, even back nearly to the occipital lobe, widespread discoloration and numerous punctate hemorrhages and apparently freshly softened, necrotic brain tissue were found. In the posterior horns of the ventricles, the lesion was limited to the tissue about the lateral wall, while the medial wall was barely affected. In the anterior horn, near its extremity, the same hemorrhages appeared all around the ventricles. In a block that passed just behind the corpus callosum, both veins of Galen were found distended and plugged by thrombi. It seemed clear that the lesion represented a hemorrhagic infarction due to closure of these veins.

Dissection of the torcular region disclosed a plexiform arrangement of the sinus system with the longitudinal sinus dividing into two unequal divisions emptying into the lateral sinuses, the larger one on the right. The straight sinus emptied as a single channel into the left lateral sinus, which was thrombosed. The longitudinal and right lateral sinuses showed a partial obstruction. The lack of any definite lesion in the cortex and in a rather wide zone of underlying white

matter is probably to be explained by the presence of this incomplete thrombosis.

3. Ipsilateral Type: In this type the longitudinal sinus runs to one side, usually the right, and the straight sinus to the opposite side. Circulation between the lateral sinuses, if present, must take place through an additional direct channel or through the formation of a foramen, both types varying con-

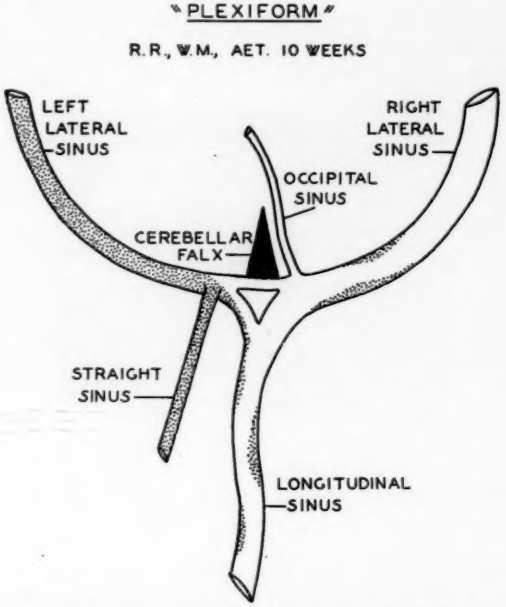

Fig. 3. Clinical illustration II. Thrombosis of left lateral and straight sinuses in "plexiform" sinus pattern.

siderably in size. In the majority of cases the lateral sinus on the side of the longitudinal sinus flow will be much the larger. This type of sinus pattern was noted in 31 preparations and was viewed with particular interest because of the character of its intersinus communication at the torcular. The longitudinal sinus entered the right lateral sinus undivided in 26 of these specimens and the left lateral sinus in the remainder. The straight sinus naturally followed the opposite course.

The right lateral sinus was equal in volume to the left in 15 cases, slightly larger in six, twice as large in five, three times as large in one and four times as large in one. The left lateral sinus was slighty larger than the right lateral sinus in two cases and twice as large in one. In 42 per cent of the cases, then, there was a right-sided over-development, frequently of a major character.

CLINICAL ILLUSTRATION—III.

Adult white male with history of otitis media showing papilledema as the only neurological finding. Normal ven-

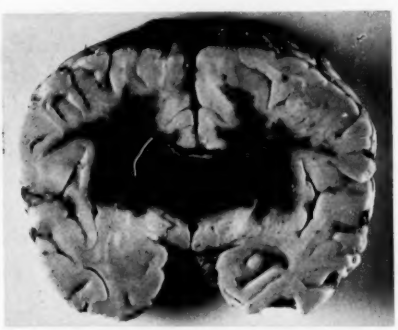

Fig. 4. Clinical illustration II. Hemorrhagic infarction of basal ganglia from retrograde straight sinus thrombosis in "plexiform" sinus pattern.

Figs. 4 and 8 are reproduced from "Disease of the Nervous System in Infancy, Childhood and Adolescence," Frank R. Ford, Charles C. Thomas, Springfield, Ill., and Baltimore, Md., 1937, by the kind permission of the publisher.

triculogram, normal ventricular fluid under increased pressure. Clinical impression of serous meningitis. Operative infection and exitus. Autopsy: Unilateral lateral sinus thrombosis in an "ipsilateral" sinus pattern.

P. C., a white man, age 46 years, U. 47,743, was admitted complaining of headache and a tender swelling on the right side of the neck. His general health had been good until the onset of an upper respiratory infection, with a sore throat, three months before admission. He had had previous sore throats without sequelae but on this occasion he developed an acute otitis media on the right and myringotomy was performed. The ear drained for 10 days. At the end of this time,

he began to complain of pain extending over the right side of the face, involving the eye, upper and lower teeth. The pain lasted about one month and then slowly disappeared. At the time of admission he was cognizant of only rare pain, localized in the eye. Two months before admission he developed a peripheral VIIth nerve palsy on the right side that cleared completely in 10 days. One month before admission a second sore throat appeared and he remained in bed for six days. He went to Florida for a rest, where for a week he fell into a marked anxiety state, was unstable, nervous, had bad dreams and night terrors, frequently waking frightened and bathed in perspiration. He had then and during the next two weeks an intermittent, dull, frontal headache. One week before admission he developed a third sore throat, and the next day a tender mass beneath the angle of the right jaw.

The temperature was 99.2°, the pulse 80, and the respirations 18. The blood pressure was 130/75. Hematological and blood chemistry studies were negative except for a leukocytosis of 15,000.

General physical study was of little importance, being marked only by a loss of subcutaneous tissue. An enlarged and tender cervical lymph node was palpable at the angle of the right mandible. The tonsils were large and chronically infected. The paranasal sinuses illuminated clearly and appeared normal. The larynx was normal. The left eardrum presented a normal coloration and normal landmarks. The right eardrum was obscured by a definite erythema and edema in the region of Shrapnell's membrane. Hearing was grossly normal.

The vision in each eye was 20/40, corrected to 20/15. The visual fields were normal for form and color, but definitely enlarged blind spots were found. The right fundus showed a papilledema of 3.5 diopters with obliteration of the neuroretinal outlines, loss of the physiological cup, overfilling of the veins and exudates about the disc. The left fundus was similar in appearance with a papilledema of 2 diopters. The remainder of the neurological examination was completely negative.

Roentgenographic examination of the base of the skull suggested a mastoid infection on the right side with extension

into the petrous pyramid. The intracranial lesion was considered to be a serous meningitis due to proximity of the infection in the petrous pyramid to the dural envelope.

A ventricular air injection was done under local anesthesia. The initial pressure was recorded as 400 mm. of water in the recumbent position. Ventricular Queckenstedt (Tobey-Ayer) test showed a rise of 100 mm. of water upon compression of each jugular vein. The ventricular fluid showed three cells and a negative Pandy test. The protein content was 25 mgm. per cent. The ventriculograms were normal, showing no dilatation of the ventricular system.

Three days later a right subtemporal decompression was done for relief of pressure. The brain was edematous, with an excessive amount of subarachnoid fluid. This procedure was followed by right simple mastoidectomy. The right lateral sinus was exposed and considered normal. The cells of the mastoid contained granulation tissue.

The immediate postoperative course was benign and on the sixth day fundus examination disclosed a definite regression of the papilledema. The decompression defect was tense. On the next day clinical evidence of a meningitis developed rapidly and exitus occurred within 24 hours. Pneumococcus type III was recovered from the spinal fluid.

Pathological Note: The superior petrosal sinus, the transverse sinus on the right side, the torcular Herophili and the superior longitudinal sinus all contained thrombi. For the most part these were tightly adherent to the wall of the vessels and were partly organized. The thrombus in the right lateral sinus was grayish-red and more or less uniform in appearance. In the superior longitudinal sinus the thrombi varied in appearance from place to place. In the posterior part there were areas which were hard, solid and opaque. Such areas alternated with what were apparently more recent clots and grayish-red thrombi. The sinuses on the left side were patent and contained blood.

On the inferior, posterior and mesial parts of the petrous portion of the right temporal bone there was a horizontal perforation, 1 cm. in length. The margins were jagged. A probe passed into this perforation communicated with the mastoidectomy wound. The dura over this was intact. The

base of the petrous pyramid had a curious purplish-red coloration. Over the mastoid cells there was, in addition, a good deal of yellow opacity. On section made into the mastoid cells, firm, yellowish tissue was noted. This was rubbery and probably represented granulation tissue.

It seemed very probable in this case that the sinus thrombosis developed in connection with the mastoiditis and that the bilateral edema of the optic discs and the increase in pres-

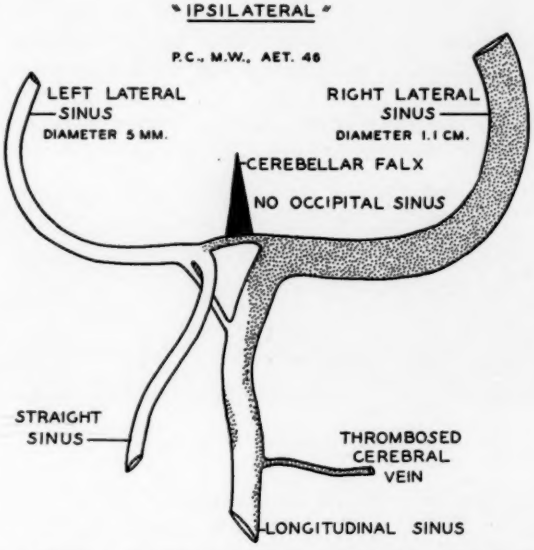

Fig. 5. Clinical illustration III. Otitic thrombosis of right lateral sinus with impairment of longitudinal sinus drainage in "ipsilateral" sinus patterns.

sure of the cerebrospinal fluid were dependent upon the thrombosis of the venous sinuses. The fresh meningitis probably arose in connection with infection of the decompression wound. Dissection of the venous sinus system showed an ipsilateral type of sinus pattern, with the volume of the longitudinal sinus passing into the thrombosed right lateral sinus. The straight sinus passed into the left lateral sinus. There was only a small communication between the two lateral sinuses at the torcular.

CLINICAL ILLUSTRATION—IV.

Female colored infant with chronic otitis media, probably unrelated to thrombosis of longitudinal and left lateral sinus, occurring in the course of pulmonary tuberculosis. An example of the "ipsilateral" type of sinus pattern with the longitudinal sinus entering the left lateral sinus alone, and the straight sinus the right lateral sinus. Hemorrhagic infarction of cortex.

J. K., a female colored child, age 18 months, H. L. H. 82,381, was admitted following a series of generalized convulsions. Both parents were known to have positive blood Wasserman tests. Two siblings had died of pneumonia at age 2 and 16 months, and three were living and well. The patient was the sixth child. For the two months preceding

Fig. 6. Clinical illustration III. Organized thrombosis in predominant right lateral sinus with clinical picture of diffuse cerebral venous stasis.

admission the parents had noticed a slow loss of weight and progressive anorexia. Two weeks before admission, bilateral otitis media had developed and had persisted. The child had vomited several times during this period. On the morning of admission the child had the first of four generalized convulsions.

At admission the child's temperature was 99.4°, the pulse 120, and the respirations 40. Hemoglobin was 48 per cent, red blood cell count was 3,500,000, white blood cell count was 12,000. The blood Wassermann was negative. The urine was negative. Blood smear showed a secondary anemia, with a normal differential count. The child was pale, listless and dehydrated. The anterior fontanelle was tense. McEwen's sign was positive. Both eardrums were perforated and drain-

ing purulent material. The fundi were clear. There were crackling râles in each chest, with diffuse impairment of the percussion note. Over the right upper chest the breath sounds were tubular in character. The liver edge was readily palpable. There were twitching movements observed over the left side of the body, and the left arm and leg showed some spasticity. Brudzinski's sign was present and there was a bilateral, unstained ankle clonus.

Roentgenography of the chest showed a disseminated tuberculosis, with cavitation in the right upper lobe.

Lumbar puncture recorded an initial pressure of 180 mm. of water. Two cells were present in the fluid, and the Pandy was negative. Four days after admission there was a complete left hemiparesis. The fontanelle was full and bulging. Exitus occurred on the following day. The clinical impression was pulmonary tuberculosis and tuberculoma of the right cerebrum. Terminal cisternal puncture disclosed 40 W.B.C., all mononuclear in type, and 5,000 R.B.C.

Pathological Note: The meninges at the base of the brain showed no gross evidence of meningitis. Practically the whole of the right hemisphere was swollen and was blackish-red in color. The only portions which escaped were the inferior surface of the frontal lobe and the tip of the temporal lobe. Over the occipital lobe there were punctate hemorrhages. There were a few hemorrhages in the meninges over the dorsal surface of the left hemisphere. The veins draining into the longitudinal sinus were distended with clots, most of them deep red in color but a few showing mottling suggestive of thrombosis. The longitudinal and left lateral sinuses were distended with thrombi. Some of the veins over the left hemisphere were also distended and some of them suggested thrombi, but this half seemed fairly well preserved. On section the dorsal and lateral half of the right hemisphere showed great swelling and edema. The gray matter was translucent and there were numerous punctate hemorrhages. In a few places the tissue appeared actually necrotic. There was, in addition to the changes in the brain, rather diffuse hemorrhage into the sulci. The tiny vessels in the left hemisphere were injected, but on the whole this portion of the brain appeared fairly normal.

Dissection of the torcular herophili disclosed the longitudinal sinus directing its potential volume into the left lateral

sinus, with the unthrombosed straight sinus entering the patent right lateral sinus. There was no cross-channel between the lateral sinuses, an anatomic abnormality which has been encountered in 8 per cent of the "ipsilateral" type of sinus pattern.

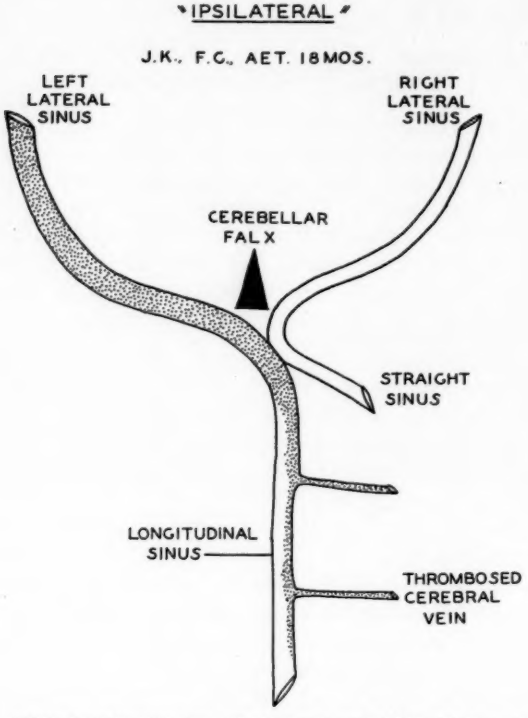

Fig. 7. Clinical illustration IV. Thrombosis of left lateral sinus and longitudinal sinus in "ipsilateral" sinus pattern.

The channels of intercommunication of the lateral sinuses in this type of sinus pattern at the torcular were of two types, and in no instance were both methods of cross-circulation seen in the same preparation. The first consisted of an oval foramen, sometimes slit-like, connecting the longitudinal and the straight sinus at the torcular as they veered in opposite directions to enter the lateral sinuses. In only one case was this

foramen so minute as to be without value as a practical means of cross-circulation. Three foramina were small, measuring from 2 to 3 mm. in diameter, and four were large, measuring from 3 to 6 mm. in diameter.

The second type of cross-circulation seen at the torcular was that of an individual venous channel, uniformly short, the longest measuring 1.3 cm. in length. In 10 preparations this single channel was large, measuring from 3 to 6 mm. in diameter (single in the majority of cases, in two preparations this channel was duplicated); in four it was small, measuring from 2 to 3 mm. in diameter; in seven it was

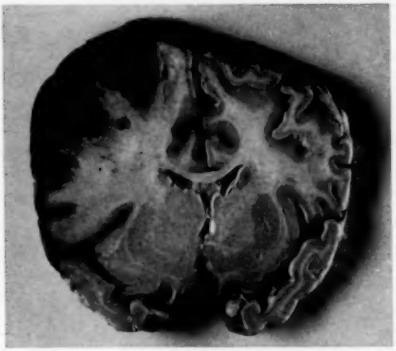

Fig. 8. Clinical illustration IV. Hemorrhagic infarction of cortex from retrograde(?) thrombosis of longitudinal sinus in "ipsilateral" sinus pattern.

minute, scarcely measurable, and in one all intersinus communication was absent. The markings of the skull were of equal intensity in 15 cases, disproportionate in 14 and indefinite in two.

The ipsilateral type of conformation of the sinuses shows an increasing percentage of right-sided predominating patterns, with the disproportion increasing in extent as compared with the common pool and plexiform types. Lack of an adequate cross-circulation was noted in eight, or 25 per cent, of the preparations in this group. Henrici and Kikuchi[18] commented on three cases in which the intercommunication at the torcular was very small, one perhaps lacking, and mentioned 11 in which it was large, attributing little significance

to the finding. In the 18 cases in which Edwards[22] noted the ipsilateral type of conformation, he found "sometimes very little intercommunication." Klestadt[24] stated that lack of intercommunication between the lateral sinuses is a factor in some cases of cerebral back pressure in the course of thrombosis of a lateral sinus. Finally, Pfeiffer[21] described one case in which intersinus circulation was effected by a paired occipital sinus of small calibre. With these exceptions, scant attention has been paid to this important variation of the torcular.

4. Unilateral Type: In this extremely rare conformation both the longitudinal and the straight sinus empty into one

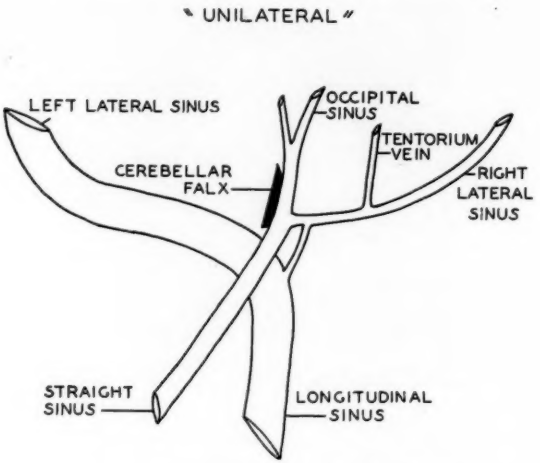

Fig. 9. "Unilateral" type of sinus pattern.

lateral sinus. The opposite sinus may be attenuated, being fed only by minute venous channels in the adjacent dura, or it may be completely absent. There were found four examples of this rare conformation of the sinuses in the 100 dural preparations studied. In three, both the longitudinal and the straight sinus entered the left lateral sinus, with the absence of a true right lateral sinus; in one, both major sinuses entered the right lateral sinus. In one of the unilateral patterns there was superimposed a gross occipital abnormality. A clinical illustration of this type of sinus pattern is described

in a later part of this paper. Two recent reports of similar variations of pertinent clinical interest have been made by Helding[25] and Hoople.[26]

In all the aforementioned general variations the occipital sinus plays a minor rôle in the formation of the variation and in the task of conveying the outflowing volume of blood. It may be absent or extremely small.

5. Occipital Type: The last main type of variation of the sinuses is directly concerned with the occipital sinus when it

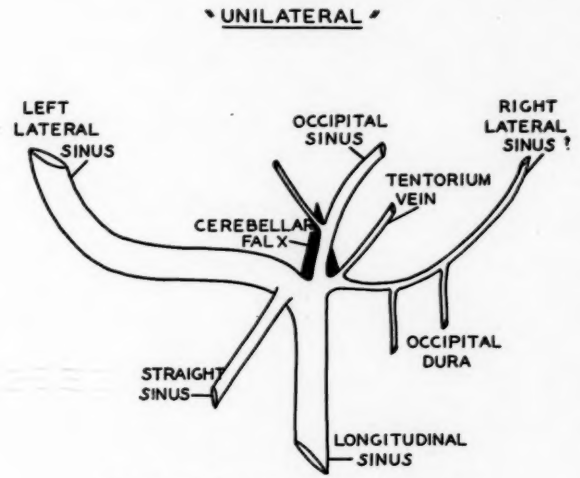

Fig. 10. "Unilateral" type of sinus pattern.

is of sufficient calibre to be of value in carrying blood. The commonest variation in the adult is the persistence of a large single or paried occipital sinus with large marginal sinuses, comparable to the pattern that is so common in the fetus or during the first few years of life. The second rare occipital abnormality is that in which either the longitudinal or the straight sinus, or both, in a common pool formation empty directly into a large occipital sinus at the expense of one lateral sinus, which will be found to be attenuated. There were two general types of abnormality of the occipital sinuses noted in this series, the first being a preservation of the fetal pat-

tern in the adult sinus system, and the second an exaggeration of the occipital sinus and one marginal sinus, connecting either with the torcular or with either major sinus and aiding materially in the distribution of the volume of blood.

There were two instances of the first type, although less pronounced variations were frequent. Both occipital sinuses were single, dividing normally into the marginal sinuses and then emptying into the jugular foramina. Both, however, measured from 6 to 8 mm. in diameter, being as large in each preparation as the lateral sinuses.

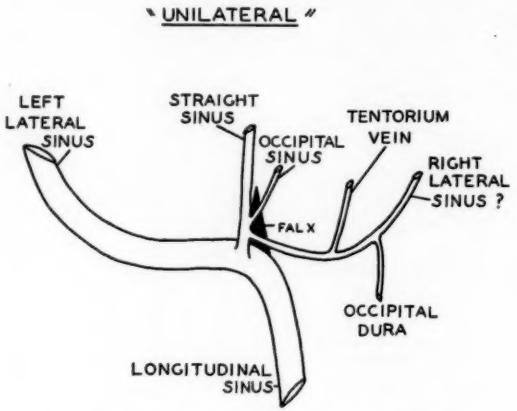

Fig. 11. "Unilateral" type of sinus pattern.

There were four instances of the second general type. One was present in an unilateral conformation of the sinuses; that is, a paired occipital sinus with one large branch extending from the left lateral sinus adjacent to the torcular to the right jugular foramen. In two cases an occipital sinus measuring from 6 to 8 mm. in diameter extended from the left lateral sinus to the left jugular foramen. Similar cases have been reported by Vernieuwe[20] and Streit,[27] in which, in addition, there was insufficient cross-circulation at the torcular. In the fourth case the straight sinus directed two-thirds of its volume to the occipital sinus and the remainder to a small lateral sinus. The longitudinal sinus emptied its entire volume into the left lateral sinus.

ABNORMALITIES OF THE LONGITUDINAL AND LATERAL SINUSES (LOCAL).

There were two instances of abnormality of the longitudinal sinus adjacent to the torcular, both being evidence of failure to complete the bilateral embryologic anastomosis along the midline of the skull. In one, the longitudinal sinus

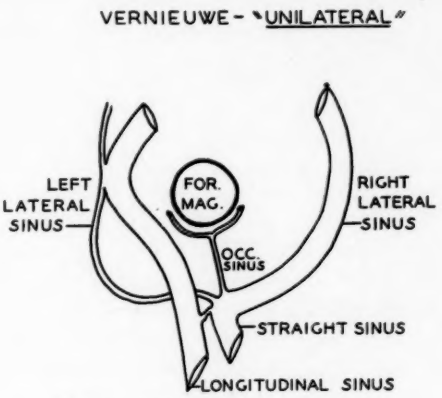

Fig. 12. Occipital sinus abnormality.

represented a double venous channel from the region of the posterior parietal lobe, about 7 cm. from the torcular. The second was a similar and shorter duplication of the sinus. starting 4 cm. from the torcular.

TABLE II.
Resumé of Abnormalities of the Sinuses.

		1. Predominance of the Lateral Sinus			2. Presence of Major Disproportion Between Lateral Sinuses			3. Presence of Inadequate Cross-Circulation at the Torcular		
Type	Cases	Right-sided Predominance	Left-sided Predominance	Equal	Cases	Right-sided Predominance	Left-sided Predominance	Small (2 to 3 Mm.)	Absent	Minute
Common pool	9	2	0	7	9	0	0	0	0	0
Plexiform	56	23	7	26	56	8	4	7	0	0
Ipsilateral	31	13	3	15	31	7	1	7	8	1
Unilateral	4	1	3	0	4	1	3	0	0	0
		39%	13%	48%		16%	8%	14%	8%	1%

Two abnormalities of the body of the normal lateral sinus were observed, representing "local migration" changes of embryologic growth. They were noted as small outgrowths of the sinus wall, occurring at the sigmoid curve; abnormalities that have been termed "lateral sinus cloacae" by Ruttin.[28]

From this brief description of the typical variations in sinus patterns it appears that the "normality" of the pattern

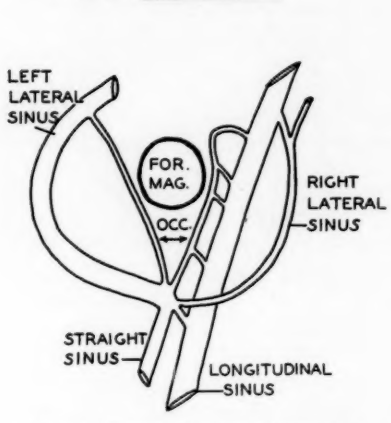

Fig. 13. Occipital sinus abnormality.

of the sinus may be more adequately defined, not by its anatomic arrangement but by the efficiency of its response to dynamic studies and to pathologic changes within that pattern. Edwards[22] has pointed out in some detail the influence of variations in the cranial venous sinuses upon the interpretation of readings of spinal fluid pressure. In a review of 150 consecutive cases at the Boston City Hospital, he noted that one-third of the readings showed differences that were clinically apparent. In one case no rise occurred on one side, *i.e.*, a positive response to the Queckenstedt test, although no abnormality could be demonstrated clinically. In a second case, pressure on the right internal jugular vein gave a rapid rise to 220 mm. of water and on release of pressure a prompt

fall to 110 mm., the resting pressure. Pressure on the opposite side gave a slow rise to a maximum of 190 mm. and an equally slow fall to the original reading. Paraffin casts of the lateral sinuses made at autopsy disclosed a smaller sinus lumen on the left side.

Canuyt and Klotx,[29] Gaillard and Mayoux,[30] Whitehead,[31] Frenckner,[32] Stauffer,[33] Lemaitre and Aubin,[34] Kopetzky,[35] Claude, Lemache and Aubry,[36] Dandy[37] and Gardner[38] have likewise recorded variations, from various causes, from the normal or expected hearing. A positive but false unilateral Tobey-Ayer reaction on an anatomical basis has been personally observed.[39] In this instance, dissection of the cranial venous system showed both the longitudinal and the straight sinus emptying into the right lateral sinus in a "common pool" formation at the torcular. From each sinus, at a point 1 cm. from its entrance into the pool, a minute channel extended to the left side. The attenuated sinus thus formed represented in position and course the left lateral sinus. It measured 2 mm. in diameter at the jugular foramen, whereas the right lateral sinus measured 1.6 cm. in diameter. In order to verify, if possible, the reliability of Roentgenographic evidence of the volume of the lateral sinuses, studies were carried out to demonstrate the correlation between the Tobey-Ayer test and the clinically available evidence in 100 individuals.

As a part of this study it was noted that the markings on the occipital bone closely corresponded to the differences in the volume of the lateral sinuses and followed exactly the distribution of these sinuses. To recapitulate, 47 of 100 skulls showed equal markings, 32 demonstrated a noticeable difference in size, 15 showed indefinite markings on each side, two showed no bony evidence of the sinus pattern on either side, and four showed no markings on one side and very definite markings on the opposite side. This observation of the relationship between sinus volume and skull markings has been extensively noted and has previously been reviewed in detail.[39] The transposition of this static anatomical knowledge into a form suitable to serve as a clinical diagnostic aid has been attempted in only scattered instances. In our own attempt to interpret sinus volume Roentgenographically, 100 consecutive anterior-posterior views of the skull were studied.[40] Our observations are summarized in the following chart:

TABLE III.
X-Ray Variations in the Lateral Sinuses.

Sinuses	No. of Cases	Degree of Variation	No. of Cases
Bilateral and equal	46	Slight difference	8
Right lateral sinus greater than left	30	Moderate difference (2:1)	15
		Pronounced difference (2:1+)	7
Left lateral sinus greater than right	11	Slight difference	1
		Moderate difference (2:1)	5
Indistinct markings	6	Pronounced difference (2:1+)	5
Absence of markings	3		
Left lateral sinus present; no right	1		
Right lateral sinus present; no left	3		

Except for isolated instances, there are few reports in the literature pertaining to this subject. The most interesting of these is that of Ersner and Myers.[41] They discussed Roentgenographic evidence secured in the course of the study of four cases of disease of the middle ear associated with thrombosis of the lateral sinus. The Roentgenograms of the first case suggested disease of the right lateral sinus and an abnormal enlargement of the left lateral sinus, a finding that was substantiated at operation; those in the second case, one of sinus thrombosis involving the right side, revealed a larger left lateral sinus; those in the third case, one of sinus thrombosis on the right side, demonstrated a larger right lateral sinus; the Roentgenogram in the fourth case, one of petrositis on the right side, showed a larger left jugular foramen. Wanamaker[42] reported an instance of variation in the sinuses demonstrated Roentgenographically; in this case the left lateral sinus was the smaller. Campbell[43] recommended the use of the Roentgenogram to diagnose anatomic variations in the two lateral sinuses but did not pursue the subject in detail. Berg and Constans[44] studied the Roentgenographic picture in 158 cases of mastoiditis prior to operation, paying particular attention to the location of the sinus plate in relation to the mastoid cells. In 11 cases the Roentgenograms showed absence of the lateral sinus on one side; in four of these cases the absence was noted on the diseased side. Both Frenckner[32] and Dixon[45] have experimented with the injection of opaque mediums into the cranial venous sinus system, the former using monkeys and human cadavers, and the latter dogs and in a few instances human clinical material. Their efforts,

although highly laudable, have not as yet brought forth a controlled series of cases from which normal variations in the sinus pattern may be ascertained. Finally, Moniz[46] and his associates have endeavored to picture the venous drainage of the brain in the course of arterial encephalography. Their plates, taken in a lateral plane from four to 15 seconds after the intracarotid injection of 15 cc. of colloidal thorium dioxide, present an effective understanding of the patency of the sinuses. No study, however, of the normal venous patterns has been made.

Clinical evidence of the relative size of the lateral sinuses secured by the use of Roentgenograms of the skull is of value from at least three points of view. In the first place, it is of clinical value to appreciate the probable volume of the main venous channels leading from the brain in cases of thrombosis of the lateral sinus or of the jugular vein in which there are signs of intracranial pressure without evidence of cerebral abscess or purulent meningitis. In the second place, as pointed out by Courville[47] in his many contributions to this and related subjects, not only simple venous stasis but actual hemorrhagic infarcts from retrograde thrombosis and cerebral abscess from a reversed venous blood flow may take place in the presence of the usual lateral sinus thrombosis. The anatomic pattern of the lateral sinus and the torcular must exert a direct influence upon the location of such lesions.

Finally, it has long been established that the Queckenstedt test, or the application of the Queckenstedt test by Tobey and Ayer, is influenced by the calibre of the efferent cranial venous channels, *i.e.*, the lateral sinuses and the jugular veins. A smaller channel, carrying a smaller volume of venous blood, will give a lower and slower response in the spinal manometer reading on compression of that channel and vice versa.

In order to point out further the relationship between the actual volume of a sinus and a Roentgenographic presentation of this volume, a comparison was made between the results obtained upon the performance of the Tobey-Ayer test and the Roentgenographic picture of the markings of the lateral sinuses upon the occipital bone. This procedure was carried out upon 100 normal individuals, and the results are assembled in the following chart:[48]

TABLE IV.
Correlation Between X-Ray and Tobey-Ayer Test.

Roentgenograms	No.	Tobey-Ayer Tests	No.
Right lateral sinus greater than left	22	Response of right internal jugular vein greater than that of left	29
Right lateral sinus visible; no left	5	Response of right internal jugular vein present; no response of left	4
Left lateral sinus greater than right	14	Response of left internal jugular vein greater than that of right	14
Left lateral sinus visible; no right	3	Response of left internal jugular vein present; no response of right	2
Both lateral sinuses visible and of equal volume	40	Responses equal	51
Indistinct markings	16		
	100		100

It is evident, then, that except for 16 plates in which the markings were indistinct, that a fairly reliable estimate of the relative size of the lateral sinus could be obtained, a conclusion that has been previously established. When, in each individual, the plates are compared with the results of the Tobey-Ayer tests, one finds a striking uniformity, which again indicates that the Roentgenogram may be considered a reliable index of sinus volume. In the relatively few cases in which the lateral sinus markings cannot be visualized in the occipital projection, a Roentgenographic projection of the base of the skull will depict the relative size of the jugular foramina, equally valuable evidence of sinus volume.

CLINICAL ILLUSTRATION—V.

White male infant who developed increased intracranial pressure following enteritis, with bilateral papilledema, convulsions and left hemiparesis. Spinal subarachnoid pressure of 340 mmg. of water. Roentgenographic evidence of right lateral sinus predominance, positive Tobey-Ayer test right, with recovery following repeated lumbar drainages.

T. S., age 26 months, M. W. H. L. H. 97,983, was admitted with a history of the onset of a left hemiparesis and loss of consciousness of six hours' duration. He was the only child of normal parents. Birth was premature at seven and one-half months and delivery was spontaneous. Diet had been well balanced and early development normal. He had had several common colds but no severe illnesses and no operations.

Approximately three weeks before admission he developed diarrhea and vomited several times. He was first treated by an outside physician three days later, and following this the vomiting ceased. Two days later the child complained of headache. One week after the onset, the child became listless, drowsy, and again complained of head pain. Three days later he was admitted to a local hospital, where lumbar puncture disclosed a pressure of 340 mm. of water; 18 cc. of fluid were removed with a terminal pressure of 120 mm. of water. Wassermann, typhoid and paratyphoid tests were reported negative. The spinal fluid was reported clear, contained three cells and showed a trace of albumen. The child improved until the morning of admission, when he developed convulsive movements of the left side of the face and of the left arm and leg. At 12:00 noon, three hours later, the child was comatose and only the toes of the left foot moved under painful stimulation.

Upon admission the temperature was 38.5°, pulse 140, respirations 40. Hemoglobin was 77 per cent, R.B.C. 4.58, W.B.C. 8,600. The differential blood count and urine were negative.

The child was comatose, pallid, and demonstrated irregular clonic movements of the left side of the body. There was a moderate cervical rigidity. The veins over the right side of the head were distinctly prominent. There was a cracked pot sound present upon percussion of the skull. Both discs showed a papilledema of 2 diopters. The arteries were quite tortuous and the veins tortuous and engorged. Both retinae were edematous and in places this edema almost obscured the retinal vessels. The ophthalmological consultant recorded an impression of "choked discs, bilateral, from increased intracranial pressure."

The anterior-posterior Roentgenogram of the skull showed the bony plate of the right lateral sinus throughout its entire length. It was much larger than the left lateral sinus impression which was seen only at the sigmoid curve. This type of cranial sinus asymmetry, a major disproportion in size with the right lateral sinus larger than the left, has been noted in 16 per cent of anatomical specimens. The deep tendon reflexes on the left side were hyperactive and the Babinski response on

the left side was positive. The clinical impression at this time was tuberculous meningitis.

Lumbar puncture demonstrated an initial pressure of 360 mm. of water, 24 mononuclear cells, and a slightly positive Pandy reaction. No acid-fast bacilli were seen in a smear and a culture was later reported as negative. The Tobey-Ayer test was not done.

A second lumbar puncture two days later showed identical findings. The spinal fluid sugar content was 42 mgm. per cent. Blood culture was negative. The lead content of the blood was 0.04 mgm./100 gm. The child's condition remained static. Two days later, the papilledema appeared less evident, the Babinski response became normal and, in general, the child seemed distinctly improved.

On July 10, a third lumbar puncture noted "less pressure," and the fluid was reported as normal. During the next four days, the child became afebrile, began to move the left extremities and began to follow a bright light with his eyes. On July 16, under avertin anesthesia to obviate all straining movements, a fourth lumbar puncture was done. There was no change in the blood pressure during the course of the anesthesia. The initial pressure was 360 mm. of water. Compression of the left internal jugular vein caused a rise of 90 mm. of water to 450 mm. When the pressure was released, the column of water fell rapidly to its former level. Compression of the right internal jugular vein caused no rise in the manometer. Thirty cubic centimetres of fluid were removed, causing a fall in pressure to 120 mm. of water. The Tobey-Ayer test remained qualitatively the same. This fluid was normal upon examination.

Rapid improvement continued, and on July 6 the spinal subarachnoid pressure was recorded as 200 mm. of water. The Tobey-Ayer test again demonstrated a block on the right side. On Aug. 1, the day of discharge, both discs showed blurring of the nasal margins and the left disc was pale, suggesting a beginning optic atrophy. There was a slight residual left hemiparesis, with a slight hyper-reflexia of the left deep tendon reflexes.

A white male child with a brief history of drowsiness, stumbling gait and convulsions, with a normal ventricular

system and normal ventricuular fluid under no increase of pressure. Positive Tobey-Ayer test obtained in the left. Clinical status of opisthotonus, generalized spasticity and tonic convulsions. Roentgenographic evidence of ipsilateral sinus pattern and clinical impression of straight sinus thrombosis. No autopsy.

E. F., a white male child, age 5 years, U. 111,979, was admitted with a history of drowsiness, stumbling gait and convulsive movements for three weeks. Birth and develop-

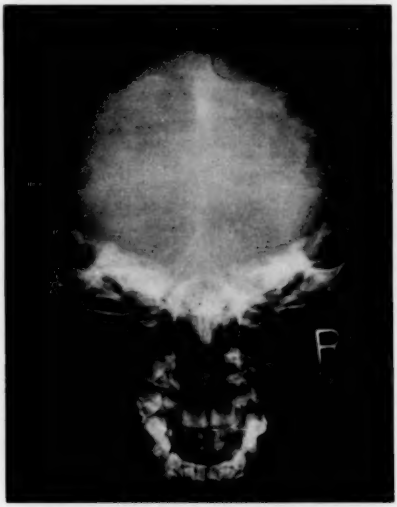

Fig. 14. Clinical illustration V. Anterior-posterior Roentgenogram of skull showing large right and small left lateral sinuses.

ment were normal. He had had dysentery at the age of 1 year, measles at the age of 2 years, and had complained of earache for several weeks at the age of 3 years. Five weeks before admission, the child was given a series of typhoid inoculations that ended three weeks ago. There were no untoward reactions to these injections. Four weeks before admission, he had stumbled and struck the back of his head, without loss of consciousness or evidence of local trauma.

Three weeks before admission, his mother noticed that the child appeared weak and restless. He spilled his food at the

table and frequently fell when he attempted to run. Spontaneous speech gradually failed and he mumbled incoherently when he attempted to talk. One week ago he became drowsy and developed a fever. At a local hospital, lumbar puncture showed three cells, and no other findings were reported. For three days before admission he had failed to respond, had been incontinent and had had jerking movements of all extremities.

Upon admission the child's temperature was 100°, pulse 140, and respirations 30. The blood pressure was 98/60. The routine laboratory examinations were normal except for a leukocystosis of 10,000. The child was semicomatose and would respond only upon painful stimuli. The head was held retracted and to the left. There was a generalized increase in muscle tone. Both arms were held in flexion, both lower extremities were extended, and there were intermittent tonic movments of the entire body. There was no papilledema. The deep tendon reflexes were slightly increased in the left leg. With these exceptions, no other findings of neurologic import were elicited.

Ventriculography disclosed clear fluid under no increased pressure (not measured). The ventriculograms were normal. The ventricular fluid showed 380 R.B.C. and 14 W.B.C. Ventricular Tobey-Ayer test showed no rise of the column of fluid upon compression of the left internal jugular vein. A second specimen of ventricular fluid showed four W.B.C. and no R.B.C. Spinal manometric readings done two days later confirmed the positive cerebral Tobey-Ayer test obtained upon compression of the left internal jugular vein. The initial pressure at this time was 80 mm. of water.

The clinical course until discharge was marked by a persistent opisthotonus, by tonic convulsions, by generalized spasticity and by coma. One month after discharge his family physician wrote: "In reply to your inquiry about this child, his condition is practically the same. He has never regained consciousness, physical condition probably not as good as when you saw him last, temperature ranges from 100° to 103° during 24 hours, still kept under sodium luminal to control his tonic convulsions." Exitus occurred two and one-half months after the onset of his illness. An autopsy could not be obtained.

Plain anterior-posterior Roentgenograms of the skull showed bilateral lateral sinus markings, with the right lateral sinus plate meeting the longitudinal sinus marking. The left lateral sinus plate stopped abruptly at the torcular herophili, strongly suggesting the ipsilateral type of sinus pattern, with the straight sinus, in this instance, draining its volume into the obstructed left lateral sinus. The clinical picture suggested a straight sinus thrombosis, similar to Cases I and II of this series.

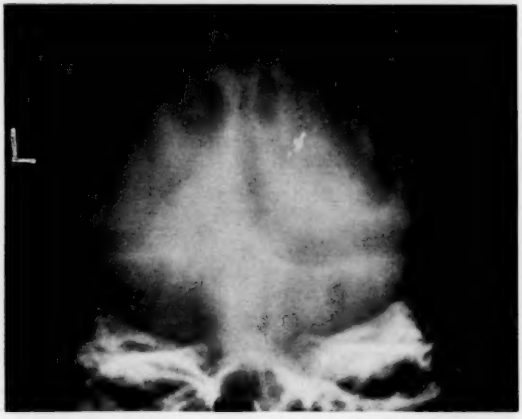

Fig. 15. Clinical illustration VI. Anterior-posterior Roentgenogram of skull showing probable "ipsilateral" type of sinus pattern.

CLINICAL ILLUSTRATION—VII.

A white male child with chronic otitis media and progressive signs of intracranial hypertension. Normal ventriculogram, normal ventricular fluid under increased pressure. Positive Tobey-Ayer response on the left side. Roentgenographic evidence of an asymmetrical sinus pattern. Relief from symptoms after ventricular drainage.

C. S., a male white child, age 6 years, U. 67,189, was admitted with a history of headache, diplopia and drowsiness over a period of four weeks. At the age of 4 years he had had a right-sided earache for one or two days, and six months later he had a similar attack of pain. He had had measles at

the age of 5 years. His mother had always considered him underweight and undernourished.

Five weeks before admission he developed a common cold. While recovering from this infection he complained of pain in his right ear. Four days later he complained of a similar pain in his left ear. The right-sided pain was more severe and continued for about a week. Neither ear was examined and neither ear drained spontaneously. Twelve days before admission he first complained of right frontal headache, sometimes radiating to the left frontal region. That evening he vomited five times. The headache persisted and he had one other episode of vomiting before admission. His mother thought that he had a high fever at the beginning of his illness, but there was no history of actual chills. He complained of constant vertigo. Four days before admission his mother noticed that his eyes crossed when he attempted to look toward the right. He had been somewhat drowsy during his entire illness, and for two weeks before admission he had remained in bed, sleeping much of the time.

Upon admission his temperature was 99.4°, his pulse 100, and his respirations 20. Blood pressure was recorded at 100/70. There was no leukocytosis. All other laboratory studies were within normal limits. He did not appear ill but was apathetic and definitely difficult to arouse. There was a suggestive "cracked pot" sound upon percussion of the skull.

Both eardrums were thickened, the right more than the left. Injected vessels were present, ranging from the periphery to the center of the right drum. Neither eardrum was bulging and there was a clear-cut light reflex on each side. There was slight tenderness over the tip of the right mastoid. Hearing in the left ear was normal; there was a mild degree of conduction deafness on the right. Roentgenography of the right mastoid showed considerable adsorption of the trabecular calcium. There was no evidence of petrous pyramid involvement.

Vision in each eye was 20/20. Visual fields were normal, although unfortunately the blind spots were not charted. The right eye converged about 10°. There was only about 150° abduction of this eye. Other extra ocular movements were normal. Diplopia fields were typical of right external rectus

palsy. Externally, the left eye was normal. Fundus examination disclosed a bilateral papilledema of 1 to 1.5 diopters, typical of increased intracranial pressure. There were no other neurological findings of pertinent interest. Ventriculography was advised, with the clinical impression of brain abscess.

The right ventricle was tapped through posterior trephine openings. Clear fluid was obtained, "probably under pressure," although no direct measurement was made. About

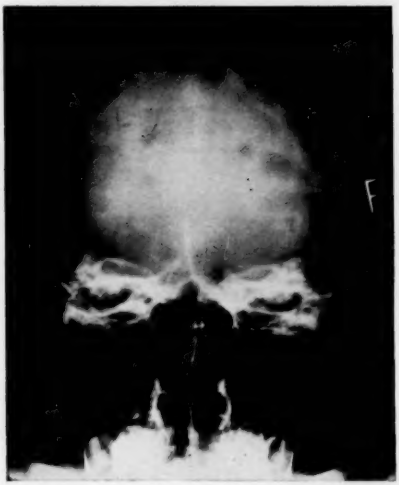

Fig. 16. Clinical illustration VII. Anterior-posterior Roentgenogram of the skull showing probable "ipsilateral" sinus pattern.

40 cc. of fluid were evacuated and replaced by air. This fluid was microscopically bloody from operative trauma. The ventriculograms were negative.

Following this ventricular drainage, his symptoms improved with astonish rapidity. Two further drainages of the subarachnoid space were made by the lumbar route and on each occasion a positive Tobey-Ayer test was present on the left side upon compression of the left internal jugular vein. Examination of the fluid on each drainage showed six cells, and 25 mgm. per cent protein content. The papilledema disappeared at the end of six weeks and he has remained well until the present writing.

Anterior-posterior Roentgenograms of the skull showed a large right lateral sinus and a small left lateral sinus with a torcular bony pattern similar to that of the preceding case.

Comment: The literature describing the syndromes of venous stasis in the brain, retrograde thrombosis, allied disturbances, such as otitic hydrocephalus, pseudobrain tumor, serous meningitis and chronic arachnoiditis, is too voluminous to be discussed in this paper. The interested reader is referred to recent review studies by Williams[49] and Symonds.[50] Obviously

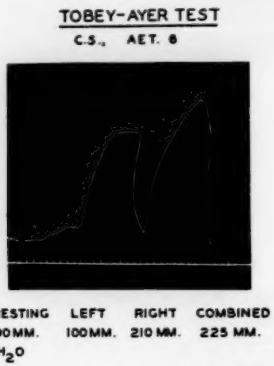

Fig. 17. Clinical illustration VII. Graph of positive left Tobey-Ayer test in patient with cerebral venous stasis.

from the clinical illustrations included in this report, thrombosis occurring in lateral sinus patterns of certain types is capable of producing either generalized intracranial pressure or, through retrograde extension of the thrombosis, a hemorrhagic infarction of cerebral tissue. The same anatomic variations will strongly influence the correct interpretation of the Tobey-Ayer test as measure of lateral sinus thrombosis. Clinically, such variations in sinus anatomy may be distinguished in many instances by Roentgenography of the skull.

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DISCUSSION.

STACY, R. GUILD, PH.D. (Baltimore): I have had the privilege of reading Dr. Woodhall's complete manuscript, and in my opinion he has handled the topic in the ideal manner. To a thorough knowledge of the literature he has added the first-hand knowledge gained by a special study of over 100 specimens. The result is a truly scholarly treatment of the subject of variations of the lateral sinus and their significance. Only if you will permit me to extend the boundary of Dr. Woodhall's topic to include the jugular bulb can I add from first-hand knowledge anything of interest to what he has presented.

Even then, I can but show you material illustrative of facts long since recorded in otologic literature; however, our textbooks give but little attention to these facts, and I believe many physicians are not aware of the common variations of the relations of the dome of the jugular bulb to the middle ear. The potential clinical and pathologic significance of each anatomic variation or anomaly, shown by the lantern slides, will be recognized immediately by the members of this audience; therefore, I shall confine my remarks for the most part to a presentation of the anatomic facts.

All of the illustrations are photomicrographs of sections cut in the so-called vertical plane of the petrous part of the temporal bone. All but the first two are from adults. They illustrate the following points:

The distance between the floor of the middle ear and the top of the jugular bulb varies greatly. Frequently a high jugular bulb is associated with dehiscences of the bone of the inferior wall of the middle ear. The bony defect may be so large that almost all the mucosa of the hypotympanum rests directly on the dome of the jugular bulb, or there may be one or more small openings. Frequently, when the bony defects are small, gland-like crypts of

mucosa grow out through them and extend for considerable distances between the bone and the wall of the jugular bulb. The lumen of each crypt is continuous with the middle ear cavity, and usually the distal part of the lumen is larger than the proximal. It may well be that many cases regarded as primary thrombosis of the lateral sinus really originate from infection of such crypts.

Of rare occurrence are the anatomic conditions illustrated in the last two slides, which show, respectively, attachment of the inferior margin of the tympanic membrane to the wall of a high jugular bulb, and a defect in the wall of the ampulla of the posterior semicircular canal. Neither patient had the clinical symptoms one might expect such anomalies to produce.

HEAD NOISES AND DEAFNESS: PERIPHERAL AND CENTRAL.*†

DR. EDMUND PRINCE FOWLER, New York.

Head noises and deafness are closely associated. Their incidence, coincidence, severity and prognosis vary greatly with the age of the patient and the type of ear disturbance. Most of the local lesions and many of the systemic disorders causing deafness are credited also with causing tinnitus. In the presence of tinnitus there is always some deafness, but this does not mean that the lesions causing these two symptoms are necessarily identical, although usually this is the case. Tinnitus is a warning signal of increasing deafness ahead; also an indication that there is still life in the neural mechanism of hearing.

Everyone notices various head noises from time to time, usually without sensing any effect on the hearing. Even in a soundproof room a "high normal" ear is capable of hearing a faint sound like that from myriads of insects in the distance, which, as Fletcher has pointed out, is due to the ever-present jostling of the molecules in the surrounding air. This is a normal phenomenon, and a large factor in determining the threshold level for hearing. It is not the type of head noise with which we are here concerned.

I shall discuss at this time sustained tinnitus, by which is meant any sensation of sound emanating within the body, no matter what its duration or fluctuation, so long as it is constant in continuity, like a drumming, a musical tone or a band of frequencies. Such noises are often described as "humming, buzzing, seething, hissing steam, or air escaping, crickets, peanut whistles, insects, machinery, listening to a sea shell, etc." Measurements of their loudness show variations from a few decibels above threshold to 45 dcb. or more.

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*Read at the Seventy-second Annual Meeting of the American Otological Society, Rye, N. Y., May 22, 1939.

†A method for differentiating peripheral from central tinnitus and deafness is here described; also a method for detecting and measuring certain effects of "subaudible" and audible tinnitus.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 29, 1939.

Careful scrutiny of the data derived from audiograms engendered the thought that tinnitus should be measured as to: *1.* its loudness; *2.* its masking by applied frequencies; and *3.* its masking effect upon applied frequencies. It occurred to

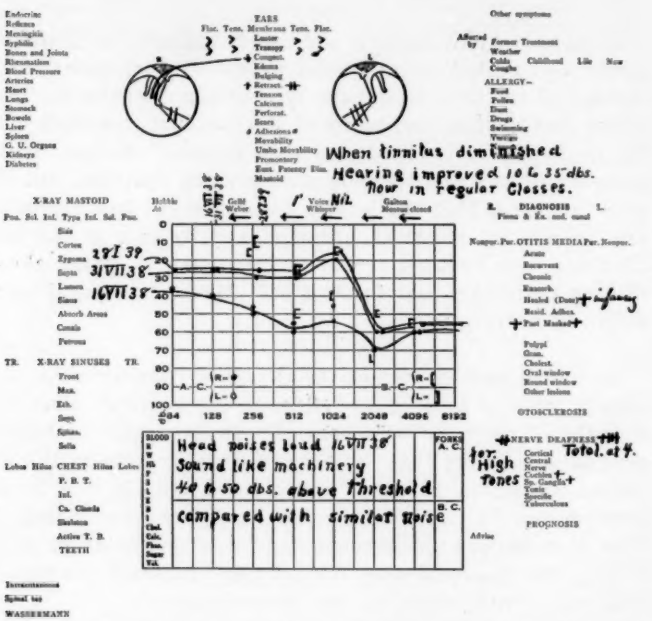


Fig. 1 shows a marked restoration of the hearing coincidental with the cessation of very loud tinnitus in the right ear. The left ear was totally deafened and the right markedly affected, following bilateral parotitis at age 18 months. Very sick with symptoms of meningitis for three weeks. The recent increase in deafness and onset of the tinnitus followed a severe psychosomatic upset. Note that the hearing improved for both air and bone conduction. Except for the improvement in hearing, no change was noticeable in the otological picture. I have many audiograms showing marked restoration of the hearing coincidental with the cessation of the tinnitus in other types of deafness. Narrow troughs may level off and the hearing improve at all frequencies by both air and bone conduction.

Following many nonoperative and operative procedures, much of the improvement in hearing may be and frequently is due to the cessation of tinnitus. This applies also to the fistulization of the labyrinth, following which cessation of the tinnitus is often one of the most striking results.

me that one might in this way obtain information as to the place of origin of tinnitus and, therefore, also of the accompanying deafness. Careful experimentation appeared to substantiate this hypothesis.

For this purpose there are available two distinct kinds of tinnitus, one vibratory, the other nonvibratory; these are usually indistinguishable to the patient:

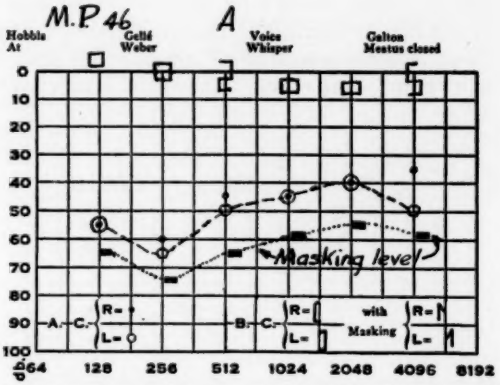
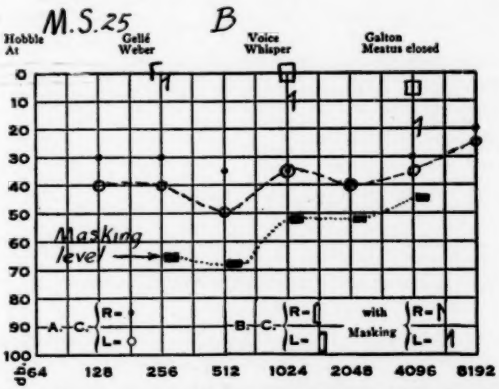


Fig. 2, A and B, show audiograms in two cases of otosclerosis. Case A suffered from a pulsating and roaring, escaping-steam tinnitus in the left ear of 10-15 dcb in loudness. This tinnitus was easily masked by frequencies 10-15 dcb. above the patient's threshold of hearing. Both the deafness and the tinnitus are of peripheral origin.



Case B complained of a constant, whistling tinnitus in the left ear which was felt as being present even when not actually heard. This tinnitus was masked by 25 dcb. over the threshold of hearing at 256 d.v., and by progressively diminishing intensities at 512, 1,024, 2,048 and 4,096. The hearing improves when the tinnitus ceases, and goes down about 10 dcb. in the frequency area of the tinnitus when it is present. Both the deafness and the tinnitus are of peripheral origin.

1. Vibratory tinnitus is caused by actual vibrations from any source reaching the end-organs in the cochlea. It may

result from direct or reflex irritations, and may originate within or be brought to the ear by way of the solid or fluid media of the body. It is always peripheral. It may occur in normal ears. It may be self-induced by various means. Vibratory tinnitus is easily masked by extraneous sounds because it is set up by actual vibrations reaching the cochlea. While present, there is some lowering of hearing, by both air conduction and bone conduction, like that observed in the

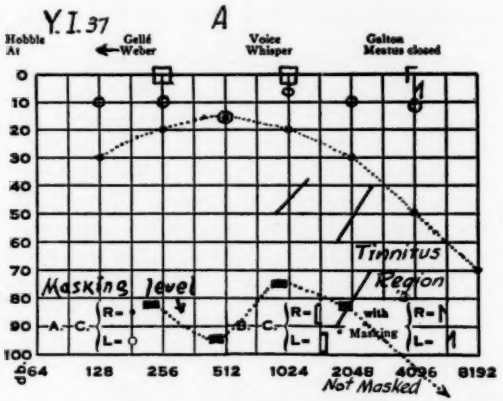


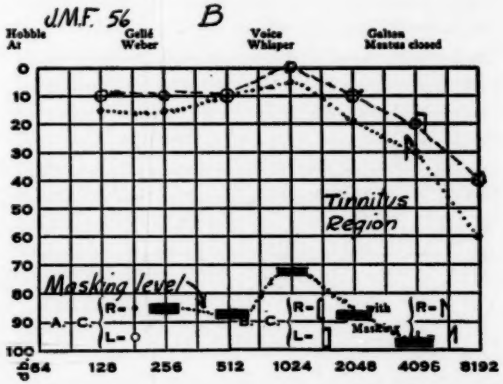
Fig. 3, A and B, show the audiograms in two cases of healed otitis with evidence of middle ear scar tissue and a marked loss of hearing for the high tones in the worse ears. In both of these patients the tinnitus was high-pitched, and masked with difficulty at all frequencies; as shown in the graphs, it required from 80-90 dcb. above normal threshold to obliterate the sound of the tinnitus. In both instances the tinnitus is, in my opinion, of central origin. The difficulty in masking is due to the fact that the tinnitus is not caused by actual vibrations in the cochlea but by some biochemical change in the central pathways. In order to eliminate the possibility of the tinnitus being heard also in the ear not being masked, binaural masking was also employed, with no change in the results.

(A) was a hyperthyroid case. At the tinnitus frequencies it was impossible to mask it. Masking noise in the opposite, normal ear also did not mask the tinnitus. At times, when the masking tones were very loud, this patient's tinnitus alternately appears and disappears.

presence of masking noise. The effect of tinnitus on bone conduction is usually ignored, although it is constant and important. Vibratory tinnitus is made up predominantly of the lower frequencies.

2. Nonvibratory tinnitus is caused by factors other than vibratory, such as biochemical stimulation. It is wholly due to lesions of the neural mechanism of hearing. It may be caused by changes of the circulation in the cochlea produced through

the superior ganglion of the sympathetic, and accompany a general vasomotor disturbance. Such disturbances are very common. Too often they are ascribed to allergy. Local pressure and blood chemistry variations anywhere along its course may initiate or increase the rate of firing of the auditory nerve. Clinically, such changes may be evidenced by tinnitus, vertigo and deafness, as in Méniére's symptom complex. Biochemical (nonvibratory) tinnitus is independent of actual vibrations in the cochlea, or anywhere else. It may be either peripheral or central. Some people can produce or alter it at will. Nonvibratory tinnitus may be made up of many com-



(B) was age 56 years, with a history of gastrointestinal disturbances. Vertigo for two years, and no deafness noticed. Both these patients gave no history of otitis media in spite of the evidence of scarred drums.

binations of frequencies, but usually predominantly of the higher pitched sounds.

Common excitants are disturbances in the gastrointestinal canal, the external and internal auditory meati, middle ear, nose, throat and teeth. Smoking and temporomandibular dysfunction are two of the culprits. I shall demonstrate by cinefluorography the jaw of a patient whose tinnitus was definitely associated with such conditions.

The lesions causing nonvibratory tinnitus activate the nerve elements actually causing the tinnitus. Any increase in its loudness is due to a greater involvement of the nerve elements and is not due to an increase in the normal stimulation pat-

terns; however, peripheral tinnitus caused by such lesions may be masked with little difficulty by vibrations near its frequency band (in the absence of middle ear impedance) because the masking sounds are aided by the increase in loudness due to the recruitment* factor always present in neural lesions.

In this connection it should be noted that high-pitched tinnitus, like applied high frequencies, requires louder sounds to

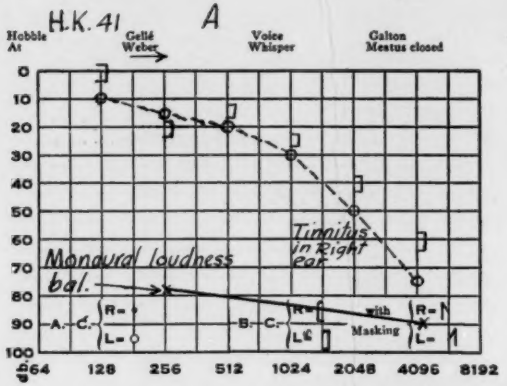


Fig. 4. A and B. These are two instances of total monaural deafness in the right and left ears, respectively. In (A) there is possibly a remnant of hearing, but no words can be understood. The tinnitus in the totally deaf right ear is 20 dcb. loud. It is masked only by the loudest buzzer or air noises when applied simultaneously to both ears. The tinnitus in the left ear is very faint (5 dcb.) and consists of a band of high frequencies. It also is masked with difficulty by either air conduction or bone conduction. Both middle ears appear normal. The air conduction and bone conduction in the left ear practically coincide, and both have gone down gradually during the past 15 years. In masking ears such as these, it must be remembered that sounds that are only 5-10 dcb. above the high-frequency thresholds are, because of the recruitment effect in nerve-deafened ears, nearly or quite as loud as in a normal ear.

mask it than low-pitched tinnitus and applied low frequencies, because it arises from a greater relative concentration of stimulation upon a smaller patch of elements than the lower-pitched sounds.

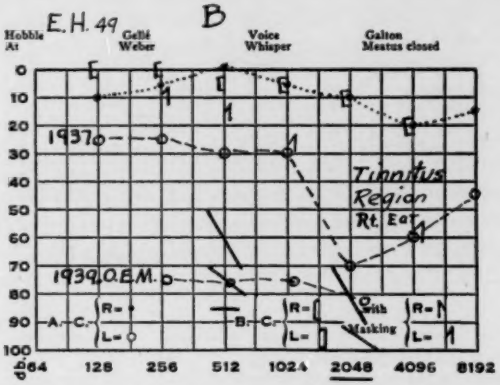
All tinnitus must be studied in relation to the following modifying factors as measured by the:

*By the alternate binaural loudness balance method it was discovered (E. P. Fowler, 1928) that an ear with nerve deafness hears the loudness of the testing sound better and better as the volume is increased. It is believed that this is because the neural mechanism is able to bring into action an increasing percentage of collateral auditory fibrils with each increase in intensity of vibration. The number approaches closer and closer to the normal number until, with very loud sounds, it has recruited enough to make the stimulation appear as loud as to a normal ear. This phenomenon I call "recruitment of loudness."

1. Excitability level of the nervous system threshold or body reflexes and of psychic disturbances.

2. Type of the underlying factors.

3. Presence and amount of recruitment of loudness with intensity.



In Case B the hearing in the right ear has always been near normal. In 1937 there appeared to be only 30 dcb. of deafness in the left ear up to 1,024; above which a marked valley or trough occurred in the threshold level, and both the binaural balance test and the bone conduction losses indicated at this time in this region a severe nerve deafness; however, the patient could not distinguish words spoken loudly into this ear. This phenomenon is very common and indicates in this case central lesions. Two years later (1939) this ear appears to be totally deaf; no sounds of any kind ear is completely masked. The plotting on the chart is only a shadow plotting due to inefficient masking. The tinnitus for the past two years has remained at about the same frequency band, and is now heard only in the totally deaf left ear. Neither discrete frequencies nor broad bands of frequencies at loudest intensities mask this tinnitus. The tinnitus in both of these patients appears to be of central origin.

Both the auditory and the static labyrinths in these two patients are now practically nonfunctioning. The deafness is probably of mixed labyrinthine and central origin. It would appear, however, that the lesions causing the tinnitus are now central. Tinnitus referred to a totally deaf ear in itself suggests a central origin.

4. If vibratory, the dominant mode of conduction to the cochlea (air or tissue conduction).

5. The summation and masking effects from or on a tinnitus of differing frequency band coincidentally present (vibratory or nonvibratory).

Central tinnitus is masked with difficulty because the centrally projected pattern is different from that of a peripheral stimulation. It does not conform to normal neural response;

moreover, nerve impulses set up in the end-organs (and, therefore, masking sounds) cannot get through; they are stopped by the central lesions. One or more links in the neural pathways are missing, and there are left few, if any, perfect

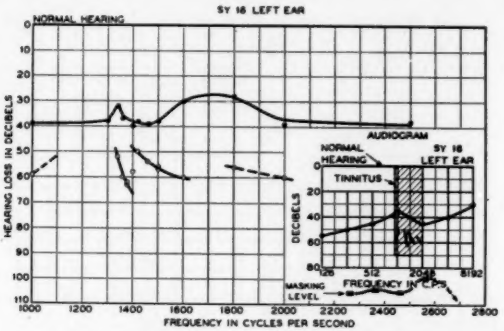


Fig. 5 shows the audiogram of the left ear of a patient with bilateral otosclerosis, uncomplicated by nerve deafness. Bone conduction was normal for both ears, but lower than is often found in otosclerosis; particularly at the lower frequencies. Tinnitus was heard only in the left ear, and in spite of its low intensity was masked with difficulty (50-60 dcb. above patient's threshold). It would appear to be at least in part a central (nonvibratory) tinnitus. For five years, off and on, it has been a humming, roaring and whistling sensations. The small area between the 1,024 and 2,048 ordinates was explored by the use of pure tones. The short lines between the 50 and 60 dcb. lines show the intensities in decibels (in relation to the normal and the patient's threshold of hearing) at which the applied pure tones within this frequency band appeared to the patient to change in character from discordant, noise-like sound to pure tone. In other words, the points at which it was necessary to elevate the intensities before they were sensed as in a normal ear.

The effect of tinnitus on the superimposed frequencies occurred not only when the tinnitus was present but also when it was absent. This suggested the exciting possibility of using this method to detect the presence of certain forms of tinnitus before they rise to the surface of consciousness; before the lesions common to the tinnitus and the threatening deafness are sufficiently extensive to cause clinical symptoms (tinnitus and deafness).

Few fibres need be firing to produce very faint (or inaudible) tinnitus; too few to produce any noticeable deafness. But the hearing will be knocked down progressively with an increase in the number of fibres engaged and their rate of firing. It is, therefore, important to stop the disturbance before many fibres become heavily engaged and the casualties mount. It would seem worth while to pursue this research further.

The upper graph will be discussed more at length by Mr. Wegel.

detours. In severe obstructive deafness, tinnitus is often heard even in a fairly noisy place. Obstructive lesions protect the ear from extraneous (air conduction) masking noise. There is no such protection in pure nerve deafness. Loud noises, of course, will distract the attention from, and aid in

ignoring, all types of tinnitus. All this has to do with the effect or lack of effect of masking sounds on tinnitus.

There is also an effect from tinnitus on masking sounds. Mr. R. L. Wegel (1931) discussed this as it occurred in one case. I have found it clearly illustrated in one of my patients. This patient noticed great difficulty in determining by voice sounds which one in a group was speaking to him. The voice appeared unnatural, as compared with a voice heard by itself. Mr. Wegel kindly retested this patient, and confirmed my findings. On repeated trials the observational differences varied only 1 to 2 dcb. To conserve time I shall discuss all these observations in connection with audiograms illustrating the various types of deafness in which they were found.

The interpretations of the neurophysiology, pathology and physics I leave to Dr. Lorente de Nó and Mr. R. L. Wegel, to both of whom I am greatly indebted for their generous donation of time and their sympathetic co-operation in the work herein briefly reported.

CONCLUSIONS.

1. There are basically two types of tinnitus: vibratory and nonvibratory.

2. Vibratory tinnitus is peripheral (direct or indirect).

3. Nonvibratory tinnitus may be peripheral or central. Central tinnitus is masked with greater difficulty than peripheral tinnitus. An explanation of why this is so is furnished by studies of the normal and pathologic neural physiology.

4. Tinnitus may be used as a means for differentiating between central and peripheral tinnitus and deafness.

5. Both "subaudible" and audible stimulation may affect the hearing for applied frequencies. Such effects suggest the possibility of their use for discovering potential tinnitus and deafness before these symptoms are noticed by the patient.

6. These studies have an important bearing on the etiology diagnosis, prevention and treatment of the disorders causing deafness.

140 East 54th Street.

DISCUSSION.

DR. LORENTE DE NO (New York): One of the reasons why I stopped doing otological work was because patients would come to me and say, "Doctor, I am not actually deaf. The thing is that I have a terrific noise in my ear sometimes, and when that noise stops, I hear well. Could you do something about that noise?"

There is not very much to be done, and, not only that, at that time it was not possible to understand why a patient will have a noise and why that noise will deafen him, and when the noise disappears the patient hears again.

If I can today discuss Dr. Fowler's paper, it is because in the past few years neurophysiology has made advances of sufficient importance to make it possible now to give an explanation how tinnitus is produced.

This explanation is not based on work of my own and, therefore, I can praise that work very highly. The explanation is sufficient, but it is possible that it is not the only one.

It was about 1931 or 1932 that a Prof. Alien, of Cambridge, made the observation that when a nerve is cut, the cut end of the nerve is the source of a continuous stream of impulses, entirely identical to those which travel through the nerve under normal conditions, which is physiologically stimulated.

That injury discharge which may be maintained for a long time would explain things like pain, and so forth.

A few years later, Dr. Gasser, Director of the Rockefeller Institute, made a fundamental observation that that injury discharge could be controlled by chemical means, but it also is controlled by changes of potential which may be recorded from the nerves.

(Slide) You will notice in this slide that the nerve has now been taken out and placed in a bath, and all the impulses and potential changes are recorded electrically. You will notice that after the nerve has discharged an impulse which is a very high potential and not included in these records, slow changes take place, which may last for a tenth of a second or even a second, or longer.

When those potentials take the course as indicated here, then a spontaneous action of the nerve takes place. That nerve, which would ordinarily have produced one single impulse during those oscillations of potential, is producing a series of impulses of that type of injury discharge.

Working in Dr. Gasser's laboratory is a Swedish biochemist, Otto Leman, who has investigated the effect of chemical environment of the nerve upon that discharge.

(Slide) Here you will notice that this nerve producing after one stimulus, one discharge, is followed by that slow potential. Now the pH of the medium is changed; there is immediately perceptible more alkaline, and you will notice when the medium is merely alkaline it produces abnormal numbers of impulses at very high rate.

Now, a very important observation was that if the ionization of calcium was present, by adding a citrate solution in which the nerve is placed, that discharge was increased enormously.

Due to this fact, the Neurological Institute in Philadelphia, Dr. Bronk and his associates, made very fundamental observations. They produced in the nerves a small source of disturbance just by applying a drop of citrate at the point of the nerve, and the result is shown in the following slide.

(Slide) The records are taken from the whole nerve. A drop of citrate has been placed at the point, and from the end of the nerve the impulses are

re-recorded. Each sharp spike is a nerve impulse. Now, that nerve is cut down under the microscope until only one fibre remains, and the impulses are recorded for that single fibre.

You will notice that that discharge is composed of a perfectly rhythmic series of impulses at a very high rate, and continuing for hours and hours.

If the citrate is now washed off with Ringer's solution, the nerve returns to normal and does not fire any more.

(Slide) But now the state can be reached, as shown in this slide, in which the nerve, treated in that manner, is occasionally filled. These marks, the marks of time, when there would be about a second or so. But now it is stimulated, and single shocks are delivered to the nerve, and an impulse is produced.

This nerve is pretreated with citrate, and the result is that the nerve, instead of producing one impulse to each stimulus, is producing a series of impulses to each stimulus.

(Slide) This shows what happens when the calcium is removed from the fluid. These records were all made in Dr. Bronk's laboratory. This shows here the response. The first spike downward signals the moment of delivery of the stimulus. This is the response. This is the line here.

(Slide) When the ganglion and the nerve are a normal medium, one stimulus produces a response. You see the base line is, except for noise, perfectly quiet. Now the calcium is removed, and the ganglion responds all right, and the base line is still quiet. But soon the response of the nerve becomes smaller, and there is a spontaneous fire in the ganglion.

Later on, when the calcium is entirely removed, there is no response any more to the stimulation, but the ganglion is fired spontaneously. Now the calcium is restored, and everything comes to the initial estate.

In the cochlea, in the course of the acoustic nerve, for some reason or other a small sound of disturbance is produced. Then that place in the acoustic nerve will be the source of impulses. If it is a small disturbance, the only change will be that the impulse produced by the cochlea will be followed by some other force which is produced by that disturbance.

The tones will not be heard as tones; they will be noises. The patient will complain of hearing, as Dr. Fowler explained, the noises distorted, and so on. If the disturbance increases, that particular tone of the acoustic nerve will produce impulses in succession, and that will be tinnitus.

Now, as those tones of the nerve are acting spontaneously, they can be modified by impulses coming from the periphery. Therefore the tinnitus will not be touched by any tone delivered to the ear. That is what actually happened in the patient that Dr. Fowler has been reporting.

Finally, if all the fibres are treated by some chemical process, then we will have a terrific tinnitus, but the patient will be deaf.

I would like to say, in conclusion, that all of those experiments were made by changing the calcium metabolism. It is not the only thing which can be used to produce those results. Perhaps they can be accomplished by intoxicating the nerve with cyanide. There are many possible causes to produce that, so you must not believe that tinnitus is produced by calcium.

But what I do say is that the effects we have observed in the laboratory, when the nerve has been treated with calcium, in which the calcium ionization has been disturbed, is a very nice model of tinnitus and gives an explanation of what might happen in the ear.

R. L. WEGEL, PH.D. (New York): This paper on tinnitus by Dr. Fowler is the latest of an interesting series, each of which has introduced a new

viewpoint for functional tests and a new technique of measurement based on the more discriminating modern acoustical methods. Over a period of years, his work along this line is gradually bearing fruit, as everyone knows.

In studying the substance of this report on tinnitus, it appears that for the first time we have something in the nature of the quantitative objective measurement of tinnitus. Hitherto, tinnitus has been pretty much what the patient has seen fit to claim it to be. I assume that when it has been demonstrated to everyone's satisfaction that the test is objective, it will assume importance, particularly in acute or prechronic cases where hearing impairment is small.

Dr. Fowler has refrained from a detailed generalization on account of the paucity of the quantitative data. It has seemed to me, however, that a provisional subdivision of his second class of tinnitus would not be out of harmony with the spirit of research. These two divisions would be these:

First, that in which the tinnitus, or its associated cause, corrupts and obliterates the character of the perceived sound. When a pure tone, such as that of a tuning fork, is used for the test this corruption occurs only when the sound is weak and within certain pitch limits which differ from one ear to another.

The second kind is that in which the interference with the perception of sound is no more than that of an equal amount of noise to a normal ear. It is hardly to be expected that these classes — that is, these two subdivisions of Dr. Fowler's second class — will ever occur in pure form, but that each case is a mixture, with generally one predominating over the other.

It is to the first of these two classes that the more definitely quantitative data pertain. When tested with tuning forks or audiometers, the outstanding characteristics of this distortion type of tinnitus are as follows:

First, distortion or corruption of the sound perceived occurs only at the lower intensities — that is, between threshold and 30 to 40 dcb. above it.

Second, distortion is limited to a narrow frequency range — one octave in the cases so far reported.

Third, this characteristic range is different for different ears. In one case, it is between 1,000 and 2,000 cycles, and in the other, 2,000 to 4,000 cycles. There are only two such cases in which quantitative data are available.

The fourth point that characterizes this type of tinnitus is that as the intensity of the tone is increased, the point at which the noise disappears and is replaced by the sensation of a pure tone is very much sharper and better defined than the threshold of hearing.

Fifth, this tinnitus seems to be most characteristic of acute cases.

It seems to me that the outstanding contribution of Dr. Fowler's paper is the outline of quantitative objective technique of measurement of tinnitus. It is to be hoped that he will continue this study. The interpretation as a mechanism of neurophysiology and clinical applications should follow the publication of more extensive, precise data.

DR. MAX A. GOLDSTEIN (St. Louis): Would you tell us, Dr. Fowler, when you make an audiometric test of a fairly complete character in an otosclerotic case, and you determine, as you can in the majority of instances, a definite tone frequency of your tinnitus, why, after the case has gone on to a further depreciation of hearing, is there finally a complete cessation of the tinnitus?

DR. FOWLER (Closing): That is some job! In the first place, I do not think that tinnitus is ever a pure tone. I believe it is always a band of frequencies. I think that many things can change the character and intensity

of tinnitus. A great many patients are relieved of their tinnitus; by simply taking them into the test room, to test their tinnitus, the tinnitus often promptly disappears.

I suffered a severe tinnitus in my left ear after a trauma from a gun explosion some years ago. After about half an hour it disappeared, but after a few years reappeared with variations. When it came back, I thought, "Well, I am probably getting on, and have sclerotic arteries, or something else imitating the neural mechanism." So I went to the acoustic laboratory, and into a soundproof room. The minute I got there, my tinnitus absolutely disappeared. It came back later in the day, and after several trials I have been able to measure it and its masking, and so forth.

On several patients tested, the same thing happened, so it takes very little, often, to change or stop a tinnitus.

I do not know how I could answer your question any other way, Dr. Goldstein.

I am not going to use more time with this subject. However, I do wish to take two or three minutes to show you an X-ray motion picture (cine-flemograph) of the jaw joint in passive and active action.

(Motion pictures) Note particularly that even when the jaw opens extensively and forcibly, and closes firmly, the condyle does not travel up and down vertically to any great extent. The jaw swings open and shut on a broad fulcrum. In some people, the fossa is shallow, and the sliding movement more apparent. The condyle clings closely to the concavity of the glenoid cavity (separated only by the meniscus). It is pushed down only when it rides up onto the tubercle in marked extension, through the downward and backward pull of the extensor muscles.

Because of the angle of the ramus with the horizontal portion of the jaw, the latter gives the appearance of descending somewhat like the jaw in that type of ventriloquist's manikin in which the lower jaw moves without a fulcrum. The human jaw does not do this. The condyle appears to descend because the angle and ramus swing down in an arc like the movable jaw of any biting instrument; this gives the illusion that the condyle is moving vertically. Careful inspection will reveal that normally the condyle does not descend to any great extent during chewing. It does, however, rotate clockwise and counter-clockwise and about a vertical axis during grinding motions.

On the side with loss of all the molars and one bicuspid tooth, note that there is a definite slap at the end of the backward excursion into the upper and posterior areas of the glenoid cavity. The ramus rocks on a movable centre situated near the junction of the upper one-third and lower two-thirds. Its motion while chewing reminds one of the motion of the eccentric rod on some steam engines. This patient suffered from considerable discomfort and ofttimes pain about the left joint, from tinnitus, and from deafness in the tonal areas affected by the tinnitus. He refused a corrective denture, but was able to control his tinnitus within reasonable limits by desisting from a nervous habit of trying to bite on the side of the missing teeth, and by abstaining from clamping down so hard on the stem of his pipe. He suffered no symptom on the right side of his face, and no right ear deafness or tinnitus.

You see it going up and down more, now, with that chewing. You see it jam back in the sphenoid cavity. Of course, there are many theories as to how this acts, none of them very satisfactory.

I thought it would be worth while seeing this, as a novelty. Thank you!

REPORT OF UNUSUAL BACTERIAL FINDINGS IN A FATAL CASE OF CHRONIC OTITIS MEDIA WITH COMPLICATIONS.*

DR. HORACE NEWHART, Minneapolis.

The isolation of a rarely encountered, anerobic, Gram negative, nonspore-forming micro-organism of the genus Bacteroides from the blood of a patient who died of an otogenous sepsis, in order to place the findings on record, warrants a brief report of this case, with the avoidance of irrelevant details.

The number of cases in which this organism has been reported as an associated or causal factor in the field of otology is very limited, especially in the English language. Recent available literature on the subject consists of two articles with bibliographies, one by John C. Henthorne and Donald C. Beaver, the other by Henthorne, Luther Thompson and Beaver,† the latter undertaken as an extension of two previous reports on the problem of nonsporulating, anerobic bacilli — one by Thompson and Beaver (1931-1932), the other by Beaver, Henthorne and Macey (1934).

I am indebted to Dr. M. I. Smith, Director, and to the laboratory technicians of Northwestern Hospital for their interest and persistent effort in isolating the organism. One of them was familiar with the work of the above authors in the Department of Clinical Pathology of the Mayo Clinic at Rochester, Minn.

Quoting freely from the article of Thompson and Beaver, "Anerobic, nonspore-forming bacteria were described as long ago as 1898 and 1899 in various putrid and gangrenous infections as follows: In the appendix by Veillon and Zuber; of the female genital tract by Hallé; of the urinary tract by Cottet; of the ear by Rist, and of the lung by Guillemot. These have been reviewed by Rist.

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*Read at the Seventy-second Annual Meeting of the American Otological Society, Inc., Rye, N. Y., May 23, 1939.

†Jour. Bacteriol., 31:3:255-274, March, 1936.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 29, 1939.

"Castelanni and Chambers coined the generic name Bacteroides in 1919, which was adopted by the Committee of the Society of American Bacteriologists in the Manual of Determinative Bacteriology in 1923. According to the manual, the genus Bacteroides included all obligate anerobes which are rod-shaped and do not form spores. The Gram-staining characteristics and the motility are variable." A further characteristic is their pleomorphism.

Ten cases have been described from whose blood Gram negative, anerobic, nonspore-forming, rod-shaped organisms were obtained. Of these, six were the bacillus fragilis; four, bacteroides funduliformis, the two differing in their morphology. Of those in whom bacillus fragilis was found, mastoiditis occurred in three, one being complicated by pulmonary infarction, one by sinus thrombosis, and one by pulmonary gangrene. Of the three associated with mastoiditis, one died, one recovered, and the outcome of one is unknown. Of the four cases in which the bacillus funduliformis was found, one had a septic temperature following an acute attack of indigestion and a fatal liver abscess, and three were associated with ulcerative sore throat. Two of these cases proved fatal.

The lesions are believed to be not due primarily to bacteroides, but to debilitating pathological conditions, as liver abscesses and carcinomata of the rectum and bladder. Chronic middle ear and mastoid infections may well constitute conditions favorable for the secondary invasion by pathogenic bacteroides. Bacteremia due to these organisms is to be regarded as a serious complication.

"The most common lesions with which bacteroides have been reported as associated organisms are primary inflammatory conditions related to or in the nose, ear, pharynx, tonsils, urinary tract and intestinal tract."

The case which came under our observation was that of a farmer, age 32 years, referred, July 29, 1938, by a physician from a neighboring city, who supplied the following history: The patient was never ill except for a chronic left otitis media with foul-smelling discharge since his fourth year. This had been treated at various times without permanent results. After an operation in the canal (probably for polyps) he was told nothing more could be done for him.

The present illness began July 1, 1938, when he noticed that the aural discharge was bloody, and he experienced pain radiating upward from the left ear. This was transitory. July 17, he felt ill on arising and by noon he had a severe chill, accompanied by sweating and vomiting. There was no vertigo. The pain noted July 1 recurred in more severe form. A local physician was consulted without benefit. On July 24, he first consulted the physician who later referred him to us. The following five days he felt much better while he was in the hospital under observation. Pupils and ocular movements were normal; the sclerae were icteric; diplococci and staphylococci were present in the aural discharge. Hgb., 79 per cent; W.B.C., 16,000. There was possibly slight haziness of ocular discs.

Temperature, July 24 to 28, fluctuated from 98° to 103° with definite spiking on three days. Sixty grains of sulfanilamide were given daily. The attending physician made a tentative diagnosis of a possible endocranial complication and referred him to us.

July 29, patient was admitted to Northwestern Hospital in a drowsy condition, but able to respond slowly to questions.

Examination showed a moderately emaciated man, greatly tanned, with marked icterus, especially noticeable in the sclerae. The neck was definitely rigid; left mastoid tip slightly tender on pressure. Ocular movements and fundi normal. Hearing reduced in left ear to two inches for whisper. Whisper perceived at 15 feet by the right ear. Rinné + on the right, — on the left; Weber lateralized to left ear. Right tympanic membrane thickened; left thickened, perforated in posterior half, with foul-smelling, thin pus flowing from between pale granulations into canal. Kernig ++ on both sides. Abdominal tenderness in upper quadrants, with liver and spleen palpable. No vertigo, spontaneous pastpointing, nystagmus, ataxia or equilibrium disturbances to indicate cerebellar or vestibular involvement. Facial, normal.

Patient complained at times of pain in the chest and paroxysms of severe coughing with small amount of sputum. This contained many different organisms, among them one which, morphologically, was found to correspond to bacillus fragilis.

Roentgenograph showed a normally pneumatized right mastoid. The left was without evidence of any cellular development, but showed an area of lesser density indicating extensive destruction posterior to antrum. Roentgenograph of chest showed both diaphragm shadows clear. Heart shadow was slightly enlarged. Increased bronchovascular markings. No evidence of consolidation or infiltration of lungs. Heart sounds normal.

Because of the very grave prognosis, Dr. Henry L. Ulrich, of the Department of Medicine, was called in consultation to share responsibility in the medical care.

A tentative diagnosis of otitic septicemia with involvement of the liver and lungs was made. The Tobey-Ayer test was not made because of the possible danger of disturbing a suspected phlebitis of the internal jugular.

Immediate exploratory operation of the mastoid was considered but was postponed pending further observation since the neck rigidity, positive Kernig and mastoid tenderness were lessening, and disappeared within four days after admission. Spinal puncture showed no increased pressure, the yellowish fluid being clear, with a normal cell count of 5.5 per cmm., and without organisms or pus cells. (A 30-hour culture of spinal fluid showed Gram positive micrococci which the director of the laboratory regarded as a contamination.) Nonne test yielded slight cloudiness. Mastoid tenderness and head pains no longer present for several days. Icterus increased. Patient's general condition apparently improved.

Smears from the aural discharge showed many different organisms, with a preponderance of streptococci and staphylococci. Blood cultures on brain broth again showed no growth at the end of 48 hours. Subsequent reports of blood cultures made anerobically showed hemolizing colonies of long, thread-like bacilli with purple granules corresponding morphologically with bacillus fragilis.

Aug. 5, because of increasing temperature and decreased hemoglobin and erythrocyte count, an exploratory operation was performed.

Removal of the compact, eburnated cortex disclosed a large cavity, which included the enlarged antrum and extended into

the tip. The cavity was filled with a cholesteatoma. The inner wall of the cavity showed extensive destruction of the bone with wide exposure of the dura of the posterior fossa. The dura was bile-stained, greatly thickened and fibrosed, indicating the existence of an ancient, organized perisinus abscess with obliteration of the sinus. The lumen could not be found by exploratory puncture. All softened bone about the enlarged antrum and towards the tip was removed and the epitympanum freed of granulations and debris. Further operative procedures were deemed inadvisable for the time being because of the patient's shallow breathing and lowered blood pressure. It was hoped that the cleaning up of the mastoid and free drainage might remove the source of the blood stream infection.

Smears from pus in left mastoid, Gram-stained, showed long, slender, Gram negative bacilli and a few organisms of mixed genera. Cultures of this pus on blood agar and brain broth yielded diphtheroids, Gram positive cocci in clumps and Gram negative bacilli. Anerobic culture showed a slow growth of hemolyzing, Gram negative, nonsporulating organisms, identified as bacillus fragilis.

For several days following the operation, the patient's condition apparently improved, except for increased icterus and further enlargement of the liver. The temperature elevations were less, and he stated that he felt better than at any time since admission. He had a very deceptive euphoria.

In spite of repeated transfusions and the administration of sulfanilamide and intravenous glucose, the temperature after a few days again rose with wider fluctuations, reaching 104.4°, with occasional chills and sweating. At times he had chest pains and paroxysms of violent coughing with scanty sputum. The cough occasionally was accompanied with vomiting.

In the face of an impending fatal outcome, with the approval of the relatives, further exploration was made, Aug. 18. After ligation of the normal internal jugular by Dr. Frank J. Corbett, a consultant in neurosurgery, the bone over the middle fossa was extensively removed upwards and backwards, exposing the greatly thickened, thrombosed lateral sinus. A fistulous opening was found leading to a small

amount of thin, dark, foul-smelling pus. After resection of the outer wall of the sinus and curettage, suction resulted in free bleeding from above.

For 24 hours following the operation the patient's condition seemed improved. He expired, Aug. 21, after being in coma for several hours.

Autopsy by the Department of Pathology, University of Minnesota, gave the following findings:

1. Right cerebellar abscess. The suppurating ear was on the *left* side.

2. Purulent meningitis of the base.

3. Mastoidectomy (complete, no cells remaining).

4. Old thrombi of superior sagittal and superior petrosal sinuses.

5. Bronchopneumonia and interstitial pneumonia; three small abscesses of right lung.

6. Subacute splenitis.

7. Chronic fibrous osteomyelitis of the petrous portion of the temporal bone.

8. Two small abscesses were found superficially in the liver.

COMMENT.

This case is reported to record the finding of the bacillus fragilis in a case of otogenous septicemia, seldom mentioned in otologic literature.

Failure to undertake early radical exploratory surgery was due to the fact that the patient's condition was probably hopeless at the time of his admission to Northwestern Hospital. The negative spinal fluid and early subsidence of symptoms of meningeal irritation were interpreted as a probable protective meningitis. Symptoms which might have helped to recognize the petrositis and the cerebellar abscess were obscured by the patient's general condition. The euphoria, which persisted up to the last hours of the patient's illness, was highly deceptive.

It is our belief that more cases of infection with bacteroidcs would be revealed by a more general resort to anerobic cultures of relatively larger blood specimens in cases of chronic middle ear suppuration complicated with symptoms of pyemia.

527 Medical Arts Building.

DISCUSSION.

DR. JAMES G. DWYER (New York): That was one of the most important contributions that has been made in a long time. There is no doubt whatever, the moral of the story is to make anerobic cultures on chronic ears and on the blood stream.

Last night at midnight I was on the phone with Dr. Newhart. I hadn't had his paper. Accidentally, he told me on the phone it was the bacillus flagellus. I didn't know of any such organism and I stayed up until half past two looking through the latest literature, and when he told me today it was bacteria flagellus or foninulo formis, there are quite a few more in the literature than Dr. Newhart has mentioned.

Now, what is back of all of this is this: The organism in the literature is not a primary invader. It doesn't occur in a previously healthy individual. The other cases in the literature that have been reported are, for instance, following a carcinoma of the intestinal tract. This organism has been found repeatedly in the nose and throat, and it is a normal inhabitant of the intestinal tract, in a big percentage of cases, but it only gets in after the damage has been done by other organisms.

It is the same way as with the bacillus pyocyaneus. We never get that, practically speaking, as a primary organism in the ear, but we get it in an emaciated child or a child in an institution that has been neglected or a child that has osteomyelitis, and so forth.

It is interesting to relate, too, that when Dr. Chuna Kline a few years ago reported her findings in the nose and throat in ozena, she repeatedly found the bacteria adis flagellus in it.

In some of these cases today, we do the bacteriology and say we find a staphylococcus or streptococcus; we treat them, and they die or they have a brain abscess, and we get staphylococcus or streptococcus from the brain abscess. Undoubtedly, if the pus from the brain abscess or from the sinus thrombosis were examined anerobically, we would probably find some of these higher bacteria. This is exactly analogous to getting the streptococcus or staphylococcus from the lung that has an actinomycosis. The actinomycosis is the primary consideration. That is the moral of the story, as Dr. Newhart has said. These cases are gone; his was gone when he got it.

It is very interesting about the icterus, because undoubtedly what this man had was a chronic running ear, probably originally due to a staph or strep, then bone disease, granulation tissue, rundown, in spite of being tanned and so forth, developed an icterus, meaning a metastasis into the liver, two or three abscesses found afterwards. He had a bacteremia, a rundown condition, if we may use that term, from this organism. Thank you very much!

DR. NEWHART: I have nothing further to add, but I wish to thank Dr. Dwyer for his elucidation of the case. We thought it worth while reporting because of the rarity of this organism associated with ear disease.

REPORT OF THE PRESENT STATUS OF THE STANDARDIZATION OF HEARING AIDS.

DR. EDMUND P. FOWLER, New York.

I have asked Dr. Newhart to report for both of us, as representing the American Otological Society on the Committee for Hearing Aids and Audiometers of the Council on Physical Therapy of the American Medical Association, so that I might utilize my time allotment to tell you of the present status of the effort to standardize hearing aids.

Hearing aid batteries have been standardized as to size and performance. There are many which exceed these requirements. Heavy duty industrial cells are usable in hearing aids, but the flashlight types are usually not economical.

The reason that no definite standardization of hearing aids is now possible is: First, that we do not now know what is desired in performance; second, we also do not know what criteria should be used in the prescription of hearing aids.

Before much progress is possible, extensive studies must be undertaken along these lines. We must formulate a background for speech interpretation and understanding of hard-of-hearing people, its changing phases, patterns, etc.

The present aim is to produce rugged instruments of long life and high standards of workmanship; to encourage precision measurements in manufacture.

Discussions are now concerned with what kind of information is needed, and I am sure the Committee of the American Medical Association and the Subcommittee on Audiometers and Hearing Aids of the American Standards Committee will appreciate suggestions and proposals along these lines.

Please address communications to Mr. Wilbert F. Snyder, Chairman of the Subcommittee on Audiometers and Hearing Aids of the American Standards Committee, National Bureau of Standards, Washington, D. C. These problems of standardization will ultimately be referred to this Bureau.

REPORT OF THE AMERICAN OTOLOGICAL SOCIETY REPRESENTATIVES ON THE COMMITTEE ON HEARING AIDS AND AUDIOMETERS OF THE COUNCIL ON PHYSICAL THERAPY OF THE AMERICAN MEDICAL ASSOCIATION.

DR. NEWHART: Supplementing last year's report, we would note that this Committee, with the generous co-operation of the Subcommittee of the American Standards Association and the United States Bureau of Standards has continued its work during the past year.

Unexpected obstacles in the form of unsolved physical and acoustic problems have been encountered which have materially delayed progress.

The tentative specifications for minimum requirements for audiometers have been slightly modified without material detriment to quality of performance. This has made it more nearly possible for manufacturers to produce acceptable instruments at a reasonable price. To date, one audiometer of those submitted has proved acceptable. Some doubtless will be found to measure up to the present required standards after relatively slight changes in construction and will be accepted.

In selecting an audiometer from the several now available, all of which possess meritorious features but have not yet been accepted by the Council, it is recommended that the prospective purchaser consider his personal needs. The first question to decide is whether he wishes an instrument which affords selection of frequencies at a continuous sweep or at fixed intervals of one octave or less. In the last analysis this will depend on what is the final decision regarding the importance of prescribing a hearing aid by selective amplification on the basis of an audiogram showing hearing acuity at smaller intervals, as octave letter spaces.

For diagnostic purposes and the prescribing of a hearing aid, provision for determining hearing acuity by both bone and air conduction is necessary. Masking when testing both

by bone and air conduction is most desirable for accuracy in diagnosis, research and in forensic work.

Supplementary or built-in equipment for testing by amplified speech and for enabling the operator to converse readily with the severely hard-of-hearing patient is very helpful.

An important task for the Committee is the preparation of a standardized audiogram blank and uniform symbols so that one person can readily read and interpret the recorded findings of another when made on a standardized instrument.

The Committee invites suggestions concerning this work from the members of this Society. At the recent meeting of the Section on Laryngology, Rhinology and Otology of the American Medical Association in St. Louis, Dr. Hayden, Chairman of the Committee, distributed a questionnaire and a suggested audiogram blank for comment. Copies will be distributed here and it is requested that all who are interested promptly respond by answering the questionnaire. Mr. Howard A. Carter, Secretary of the Council on Physical Therapy, will tabulate the replies.

Recently the Committee has been asked to expand its field and investigate the performances of simple, pure tone audiometers now being made available for screening school and industrial groups, as distinguished from instruments for clinical purposes.

Because of the wide interest shown in the early detection of hearing deficiencies as an important step in the conservation of hearing, there is a growing demand for audiometers of this type. It is therefore important, in order to insure comparable results of scientific value, that the performance of such instruments used in screening large school and other groups be standardized.

The work of establishing minimum standards for the performance of hearing aids and checking new models, including those of the vacuum tube amplifier type with crystal microphone, is going forward consistently. Other related problems are receiving the serious consideration of the Committee.

REPORT OF CENTRAL BUREAU OF RESEARCH.

DR. EDMUND P. FOWLER, New York.

Two informal meetings of the Trustees were held during the year, and the Annual Business Meeting was held at the office of the Bureau in the New York Academy of Medicine on Saturday, Jan. 21, 1939.

On account of illness, Dr. J. Gordon Wilson, Chairman of the Board of Trustees, was unable to be present and submitted his resignation as Chairman. This resignation was accepted with deep regret and the Board expressed its gratification that Dr. Wilson still remains a Trustee.

Dr. D. Harold Walker, of Boston, was unanimously elected Chairman of the Board.

The resignation of Dr. H. S. Birkett, of Montreal, was submitted and accepted with deep regret, with due acknowledgment of long and valuable service recorded in the minutes.

Dr. Charles B. Davenport was appointed Consultant in Genetics to the Board.

Prof. J. B. Collip, of McGill University, was also elected a Consultant.

Dr. Marvin F. Jones, of New York, and Dr. D. E. S. Wishart, of Toronto, were elected Trustees of the Research Bureau, and accepted.

Mr. Starling W. Childs spoke on the state of the capital of the Bureau. Without cost to the Bureau, Mr. Childs had had prepared, and now submitted, a review of the securities. Mr. Childs made an explanatory statement, and on motion the thanks of the Bureau were extended to Mr. Childs and to Mr. J. Dupratt White, financial advisers to the Board.

The Bureau asks the aid of the members of the American Otolological Society, and through them the members of local and State Societies, to obtain reliable information concerning the onset and progress of otosclerosis in monoval twins of known otosclerotic heredity. This, of course, is in the effort to differentiate between hereditary and environmental factors.

A card has been prepared by Dr. Davenport and Dr. Fowler. Address all communications to Dr. E. P. Fowler, 2 East 103rd street, New York.

The Bureau also wishes to enlist the assistance of otologists throughout the country in obtaining accurate figures concerning the effect of single or repeated pregnancy on the onset and progress of otosclerosis.

The Bureau desires reliable statistics concerning the results of surgical interference aimed at improvement in hearing in otosclerosis. Audiograms are necessary in all of these investigations, and in the surgical cases sufficient time must be allowed to elapse before attempting to establish a reasonable end-result. The method of operation should be clearly epitomized.

Search and tabulation of the foreign and domestic literature on chronic progressive deafness still continue.

2 East 103rd Street.

REPORT OF BOARD OF TRUSTEES OF THE RESEARCH FUND.

DR. D. HAROLD WALKER, Boston.

I greatly regret that Dr. Gordon Wilson is unable to be present with us at this meeting. For this reason, it is my duty to report the activities of the grantees of the Research Fund during the past year. I shall briefly summarize the results of their investigations, which have been and are being carried on by Prof. Bast, of Madison, Wis.; Prof. Anson, of Northwestern University, Chicago; Dr. Fowler and Dr. Jones, of New York; Prof. Tait and his associates, at McGill; Dr. Davis and Dr. Lurie, at Harvard; Dr. Culler, of Rochester; Prof. Knudsen, of Los Angeles; Prof. Collip and his associates, at McGill.

Dr. Anson's report is divided into four parts: The first is concerned with the pneumatization of the petrous apex of the temporal bone, with special reference to the vascular and the pneumatic routes by which infection may spread to the labyrinthine spaces. The description of this work is based upon wax plate reconstructions. Dr. Anson has published a paper on this subject, which appeared in the *Archives of Otolaryngology* in May, 1938. Secondly, he continues to be interested in the fissula ante fenestram. Dr. Wilson mentioned the importance of this work in his report of last year. Prof. Anson states that "the tissues in the fissular area are histologically unstable, and the persisting cartilage, with potentialities of fresh growth, is genetically associated with the production of otosclerotic bone at this 'site of predilection'." Third, he has published a paper, also in the *Archives of Otolaryngology*, on the stapes. In an adult of 18 years, the vestibular aspect is almost cartilaginous; bone appears in an adult of 57 years merely as islands, which increase in size and number in older individuals. He is now engaged with Dr. Wilson in making a comparative study of the otic capsule in the lowest forms of animals.

Prof. Bast earlier studied the anatomy of the left ear of the fetus and of the child. This past year, he is engaged in similar study of the right ear. He expects to publish his paper very

soon, based on over one-half million serial resections of the petrous bones from 27 infants and young children, and 68 bones from 42 fetuses, ranging from 21 weeks to term. He is still interested in the utriculoendolymphatic valve, and states, "We have made several models from serial sections and have a pretty good concept of the structure, but the function is harder to interpret because we do not know what variations in pressure occurred in the various fluid chambers during normal life." The Committee has seen fit to help him to continue this work.

Dr. Culler has transferred his interests to the University of Rochester. His report is interesting and quite varied. First, he has been injecting a parathyroid extract called "paroidin" into young dogs. It is not necessary to describe in detail the symptoms of the effect of this injection. He has one animal which he hopes to dispatch soon, and may show some change in the cochlea. Hearing tests reveal a decrease in hearing for three different frequencies. Secondly, he has been interested in renewing Wittmaack's work on otosclerosis. You have probably read his description of how, when injecting ink into the semicircular canal of chickens, that he sometimes injured the venous system and as a result later found bony changes which were characteristic of otosclerosis. Culler hopes to be able to measure the hearing in chickens and to progress along Wittmaack's lines. He has other methods in mind to cause a venous stasis in other animals. Third, Culler has been interested in localization of losses in auditory acuity in various frequencies in the brain. He begins with the geniculate bodies, and has described in detail the various aspects of the bodies where he has obtained evidences of sound tracts. These tracts he continues to the higher centres, and intends to attempt the mapping of the cortex. His report upon this subject requires a very thorough digestion of the facts before one appreciates what he has been able to accomplish. Fourth. Culler has been interested in the X-radiation of the pituitary body in which a temporary improvement of hearing was attended by a concurrent depression of blood-sugar. These experiments seem to show that a lower sugar concentration allowed transmission of fainter sounds to the basilar membrane. He cites two illustrations, one by injecting distilled water, which caused an improvement in dogs' hearing, and the other, injection of salt solution, which was followed by a decrease in hearing. He wishes to continue these studies.

The report of Davis and Lurie shows, first, the compilation of results of quinine upon hearing. Whether these results are of any help to the otologist clinically remains to be seen. Dr. Lurie's work on waltzing guinea pigs is very interesting and may prove to be of great value. Only recently he has been able to localize areas in the brain which result in disturbances of equilibrium, and which may give us a different idea as to the cause of certain cases of vertigo. The publication of the book called, "Hearing: Its Psychology and Physiology," by Dr. Stevens and Dr. Davis is a very excellent work. It is worthy of anyone's perusal who is at all interested in the subject. The laboratory at Harvard is especially well equipped for the study of the physiology of hearing, and I am glad this work will continue. Both Davis and Lurie have plans for additional experimental work for this coming year in testing their theory that the nerve fibres are excited by the hair cells by means of a chemical mediator; and a second line of investigation, the study of electrical reactions of the cerebral cortex response to auditory stimulation. They have mapped out a very elaborate program. which will require the collaboration of several investigators over a considerable period of time.

Dr. Jones has been working on the sympathetic nervous system and reaction of labyrinthian fluids as an "allergic phenomena." He has succeeded in establishing certain basic methods for investigation of the cochlea and is now ready to proceed with his experiments. He also is continuing the collection of temporal bones and is in a position to obtain a great deal more material.

Dr. Mortimer, Dr. Wright, Dr. Thomson and Dr. Collip, of McGill University, report the result of the effect of estrogenic substances in atrophic rhinitis and ozena, and hearing. The report is a very long and detailed one, and the results are given in a summary. *1.* The results of the use of estrin in 55 cases of constitutional deafness. *2.* High frequency of occurrence of cranial dysplasia in 153 cases of constitutional deafness. *3.* It is suggested that these two affections may have a common background. *4.* It is their opinion that the therapy which acts upon nasal disease is able to produce improvements in certain individual cases of deafness. This work, to my mind, is extremely interesting and well worth further investigation.

Dr. Dworkin continues his work on the relation of the pituitary gland to general growth, and to the localization of sound appreciation in the cochlea. He also has been studying the effects of vitamin A deficiency upon hearing. He experiments upon rats, some normal, and some with the gland removed. As a result he finds that there is no change in the organ of Corti or of the nerve cells. He does find, however, that in rats where the gland is removed there is an abnormally shaped footplate of the stapes. He has been able to drill almost invisible holes into the various portions of the cochlea and to observe the result. He concludes: *1.* "When the apex of the cochlea is damaged, one may obtain total failure of hearing; but when the loss is only partial, it concerns not *pitch* primarily, but *intensity*." *2.* "When the base is damaged, high pitch perception is most markedly affected or entirely lost. The former conclusion argues against low tone localization in the apex; the latter, in favor of high tone localization in the base." He goes on to explain the fact that total failure occurs after the apex is damaged, that low tones will not be readily heard, but when the intensity is raised, harmonics are generated. The harmonics stimulate a functional portion of the organ of Corti and reproduce the fundamental tone. The results of the findings of Sir Edward Mellanby suggested the experimentation of deprivation of vitamin A. Mellanby found a destruction of the VIIIth nerve in dogs, by a diet deficient in vitamin A, and rich in cereals. Prof. Dworkin wishes to use young rats and puppies and to experiment further along these lines.

Dr. Fowler, Jr., is very fortunate to have worked with Dr. Holmgren and to have visited Dr. Sourdille. There is no question that the operation for fenestration for the relief of deafness must receive serious consideration, and the result of Dr. Fowler's experience with Lempert, Sourdille and Holmgren is of the greatest importance to us all. As he mentions in his report, the importance of an accurate test of the bone conduction is of the greatest importance and any experiments which will rectify misleading tests will be of great value. Dr. Fowler wishes to continue experiments with flaps for the fistula and also is interested in the effect of cold water as a cause of exostoses in the external auditory canal.

Dr. Fowler, Sr., is continuing his work upon hearing tests. He has observed new phenomena which may be of the greatest

assistance in diagnosis of certain cases of deafness. He is experimenting with methods of measuring loudness of tinnitus, and of a more accurate method of determining bone conduction. All this work is most important in advising the use of a hearing aid. Dr. Fowler also speaks of the importance of the proper fitting of hearing aids, and I hope he will continue this research.

Prof. Knudsen's report must be read in detail to be appreciated. He gives a summary of words used in his articulation test for hearing aids, and the total amount is over 62,000. There are 11 observers, and No. 7 is an otosclerotic. This case is used for all the tests, to the extent of 32,000 words. Uniform amplification is more efficient than uncontrolled amplification, as is given by the usual portable carbon aid. With a wearable bone conduction aid, the percentage of articulation was 72; with laboratory uniform amplification, the percentage was 97. Speech, with equal amplification of all tones, "is more intelligible than speech with loudness equalized to meet their varying deficiencies." In other cases, especially observer No. 7, selective amplification was immediately effective. This is especially true in the loss of the consonants. He cites another case, No. 1, with uniform air conduction loss in both ears and almost normal bone conduction at a level of 36 dcb. Above this threshold the percentage was uniform, 77 per cent; selective, 81 per cent. When a portable vacuum tube aid was used on observer No. 7, the syllable articulation was increased from 19 per cent with no aid to 87 per cent with a portable vacuum tube aid. This was at a distance of three feet. This statement is interesting; namely, that in a classroom, observer No. 7, sitting in the third row, without an aid, heard nothing, but by cupping his hand behind his ear, his hearing increased to 29 per cent. The increase was 34 per cent with a carbon aid, and 64 per cent with a vacuum tube aid. There is also some interesting information, of which he speaks, regarding filters, and the difference between a man's and a woman's voice. He speaks of the fact that theoretically the efficiency of bone conduction may attain the same degree of perfection as air conduction. The results of his tests with various observers do not prove his point. It is very likely that the percentage of improvement with bone conduction will be increased over air conduction with the use of a better type of instrument. The work on binaural tests

can be summarized in Dr. Knudsen's own words; namely, "These tests indicate that if binaural aids are used, and the loudness adjusted to equality in the two ears, the setting should be made for a comfortable level in the better ear, and the sound made equally loud in the poorer ear, provided the level is comfortable."

We deeply appreciate the work done by the above-named gentlemen, and the interest they have taken in our problems, especially when one considers the limited means which we are able to place at their disposal. We are delighted that they see their way clear to continue their investigations.

5 Bay State Road.